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# ALASKA MEDICINE

Volume 35, Number 1

January/February/March 1993



*Official Journal of:*

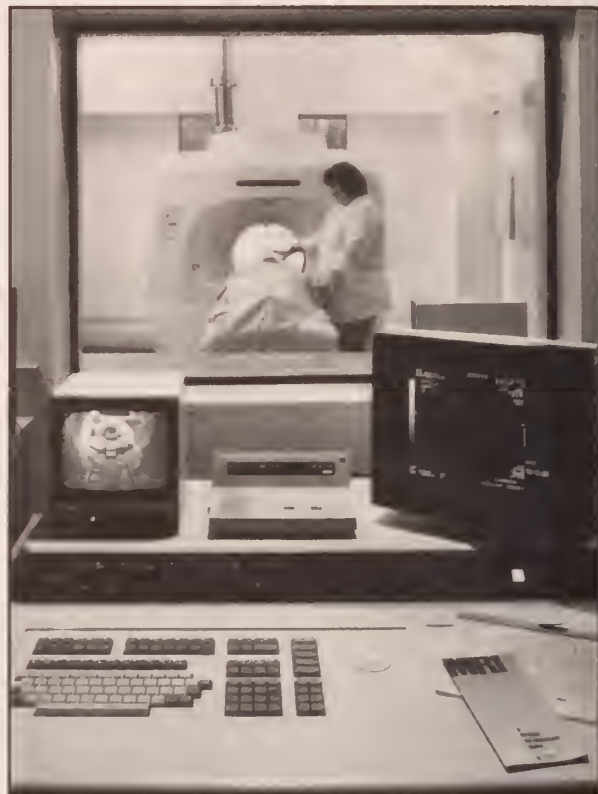
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*In this issue:* Cold Injury: A Collection of Papers by William J. Mills, M.D. and Colleagues



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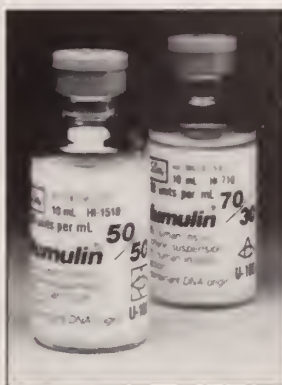




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# ALASKA MEDICINE

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ALASKA MEDICINE gratefully acknowledges the following for their monetary contributions to this issue: *Alaska Arctic Medical Research Foundation, Alaska Regional Hospital and Staff, American Society for Circumpolar Health, Robert Gottstein, Medical Arts Pharmacy, and Providence Hospital and Staff.*

**About the cover:** Classic Hand - "This hand represents a frozen extremity (superficial to deep) 24 hours after rapid rewarming in water at a temperature of 108 degrees F. (42 degrees C.). Exposure 20 minutes, ambient temperature +5 degrees F (-15 degrees C), winds 50-60 knots, Arctic Alaska. (Windchill factor near -45 degrees F. (-43 degrees C.). The large, clear, and pink blebs, extending to the digital tips, with adequate capillary filling of the nail beds, represents an excellent early prognostic sign.

# Editor's Note

It has been my privilege to have known Doctor Bill Mills for a little over a quarter of a century, during which interval I have followed with admiration, his work in thermal injury. He has become a world-renowned authority on the subject and commands appropriate respect from his colleagues. (I'm merely a pathologist, so my experience has been oblique.)

When Dr. Mills retired recently and noticed the need for a project, it occurred to us that since he had published his early papers in *Alaska Medicine* (of which he was the founding editor -- thus insuring acceptance), and that this little journal was not then listed in Index Medicus, some of his light was, and remains, hidden under the proverbial bushel. We decided to

devote an issue to his subject, to republish the original articles, and to allow him to update that body of knowledge. We also solicited an article from Dr. James O'Malley, a second generation Anchorage surgeon, Dr. Mill's protege and probable successor in the field; and from Dr. Bruno Kappes, a psychologist on the thermal injury team.

We hope you find this issue interesting historically, and useful in dealing with cold injured patients.

(S)

Donald R. Rogers, M.D.  
Editor, *Alaska Medicine*

## Letter to the Editor:

This issue of *Alaska Medicine* pays appropriate tribute to Dr. W. Mills, who is acknowledged as one of the discoverers of the present method used to successfully treat frostbite and hypothermia. I have had the pleasure of interacting with Dr. Mills ever since I met him at a hypothermia meeting held in Rhode Island in 1977. During those days, there was considerable controversy concerning various rewarming therapies, especially in the field. In the course of one of these discussions, Dr. Mills described his clinical efforts in rewarming hypothermic victims in the field, and hospital.

Dr. Mills was aware of the special problem associated with the identification and treatment of frostbite and hypothermia. He was able to draw on his vast experience as a physician/scientist and arrive at several observations. Besides his scholarly activity which is evidenced by a republication of some of his articles in this volume, he has worked unceasingly to help produce a high altitude laboratory at Mt. McKinley. This he did as Director of the Center for High Latitude Health Research, University of Alaska, Anchorage. His efforts along with those of other scientists has produced an outdoor laboratory that is constantly being used to evaluate human performance at high altitudes.

His major accomplishments in the field of hypothermia include the astute observation that hypothermia

induces a decrease in metabolic rate associated with dehydration. The "Metabolic Icebox" term used to describe this state was originated by Dr. Mills to describe the various effects of hypothermia.

In addition to being published widely and being a guest speaker at National and International meetings, he continues to be interested in the cellular effect of frostbite. He is impressive with his zeal to constantly seek various clinical or basic science clues towards predicting the effectiveness of various rewarming strategies.

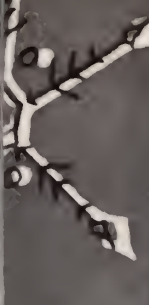
Dr. Mills in many ways popularized nationally the notion of hypothermia and established Alaska as one of the key areas to conduct cold weather/cold water research. Many frostbite/hypothermic victims have been saved by the direct or indirect efforts of Dr. Mill's research in this important area.

I have known Bill not only as a physician/scientist, but also as a friend. He symbolizes what the classic physician should be, both a scientist and a clinician with a sense of compassion.

(S)

Robert S. Pozos, Ph.D.  
Director, Thermal Physiology  
Naval Health Research Center  
San Diego, CA 92186-5122





# ALASKA MEDICINE

Volume 3, Number 2

June, 1961



## FROSTBITE:

Experience with Rapid Rewarming and Ultrasonic Therapy

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# FROSTBITE: EXPERIENCE WITH RAPID REWARMING AND ULTRASONIC THERAPY\*

WILLIAM J. MILLS, JR., M.D. and ROBERT WHALEY, M.D.

ANCHORAGE

## Part 1

### I. INTRODUCTION

Cold injury, a major medical problem of the military surgeon in time of war, is at any time a matter of concern to the physician in the Arctic and sub-Arctic areas. Cold trauma is of worldwide occurrence and encompasses the patterns of chilblains, immersion foot, trench foot and frostbite<sup>(1)</sup>. It may occur even in tropical latitudes in mountainous terrain where an arctic environment may be found, and in tropical waters after long immersion. In the event of a rare catastrophe, high altitude flight provides a setting for severe cold injuries where the complications of anoxia, itself thought to be a predisposing factor by some authors, is often present.

Reports of injury from cold may be found in most Alaska hospital records. The general experience indicates that frostbite or true freezing of tissue is the commonest form of cold injury treated in Alaska<sup>(2)</sup>. Here one naturally expects and finds this injury most common in winter, but hunters and mountain climbers have fallen victims even in July and August.

An examination of hospital records, and discussions with physicians, both civilian and military, throughout the state, have demonstrated much disagreement in methods of management of the injury. Here and elsewhere frostbite has been treated by such highly variable methods as packing the part in ice, primary application of pressure dressings, sympathetic block, insulation of the affected part at room temperature, and by the application of local heat in many forms, in-



Dr. Whaley

Dr. Mills

cluding the use of diathermy<sup>(1, 3-9)</sup>. Adjunctive measures have included sympathectomy, sympathetic block, anti-coagulants, antibiotics, vasodilators, corticosteroids and combinations of these. Early or late debridement and/or amputation has frequently been a part of these variable programs<sup>(4, 10-11)</sup>.

Some variation in treatment may be expected in a group of physicians and the individual cases may demand some adjustment of methods. It would seem, however, that the latitude of treatment permitted between packing the part in ice on the one hand and immersing it in a warm water bath on the other, or between considered watchful neglect on one hand and early amputation above the site of demarcation on the other could profitably be narrowed.

The purpose of this paper is to report our initial experiences with frostbite and to review such current opinion in the field as may be of

\*Editor's Note: This is the first of three parts; Parts 2 and 3 will be published in the June and September issues.



interest to Alaskan physicians. The interest of the authors in this problem was aroused early in 1955 by a group of patients who had sustained clinical frostbite, and all of whom had undergone amputation of one or both of the lower extremities at levels varying up to the low thigh. We were stimulated to try to find some treatment that would minimize or eliminate such severe losses.

This concept has been re-enforced after treatment of a number of cases of frostbite at the Alaska Native Hospital and in Providence Hospital in Anchorage. A series of cases will be presented in which the treatment consisted of early or delayed rewarming of the involved part, scrupulous protection of the injured extremity from trauma and infection, the avoidance of unnecessary debridement or amputation, and the early institution of physiotherapy, both active and passive, and in most cases the use of ultra high frequency sound in a water bath during the critical first three weeks. Serial serum glutamic-oxaloacetic transaminase and other enzyme determinations were used as additional objective measures of deep injury.

## II. BACKGROUND

Frostbite may be defined as the cooling of body tissue to the point of ice crystal formation<sup>(12)</sup>. There have been numerous classifications of frostbite as to severity, the duration of exposure, the type of cold (wet or dry), the rapidity of freezing and other factors. Further, it has been customary to classify the injury in "degrees," similar to the older burn terminology<sup>(1, 4, 9, 13-14)</sup>. A variety of signs are usually listed to determine the "degree" of frostbite and therefore to guide treatment. Although some differences in management will exist between more trivial and serious cases, it is apparent that, as in burns, even the experienced clinician will have great difficulty in accurately classifying the severity of injury early and that a more simple classification as to superficial or deep would probably be more suitable. Moreover, the involved extremity may exhibit several degrees of injury without regard to a regular pattern or progression. As will be discussed later, a further classification of great usefulness clinically is whether or not there is significant lowering of general body temperature.

Analogies have been drawn between frostbite and thermal burns<sup>(15)</sup>. Although both of these injuries result in blister formation, similar early microscopic changes in muscle tissue, and certain similarities in gross appearance, it is the firm opinion of the authors that this analogy is not an accurate one and is particularly undesirable because of misleading inferences often drawn therefrom.

The mechanism of injury in frostbite, although still not clearly understood, apparently depends on at least three distinct processes. The first and most obvious is the actual disruption of cellular and tissue structure due to ice crystal formation. Experimental work on laboratory animals by many investigators has demonstrated that the tissue injury is greater in conditions where cooling is slower, where the period of cold is prolonged, and particularly where the rate of rewarming is slow even for cases with roughly equivalent depth of frost penetration<sup>(15-18)</sup>. Meryman, working at The United States Naval Medical Research Institute, has done extensive pathological studies of such tissues and has demonstrated that the size of ice crystals formed in tissues is inversely proportional to the rate of freezing, and that the prolonged maintenance of a tissue in a partially frozen state where ice crystals are in equilibrium with the tissue fluid results in slow accretion to these crystals with a growth in their size and further tissue damage<sup>(19)</sup>. These observations are consistent with predictions from our knowledge of the characteristics of two-phase equilibrium mixtures in other situations and is also consistent with the experience of the meat packing industry that rapid freezing and adequately low temperature maintenance is necessary to proper preservation of food stuffs<sup>(12-20)</sup>. There appears to be good evidence from this and other work that the maintenance of a tissue at its freezing point, usually —2 to —5 degrees Centigrade, may be more detrimental than the maintenance of this tissue at a much colder temperature<sup>(12-17)</sup>.

Associated with the ice crystal formation is some type of direct cold injury to protoplasm which is probably in part at least due to extensive dehydration. This apparently is only partially reversible.

A third type of tissue injury is that due to impaired circulation. This is evidently a prominent cause of injury in "trench foot" and "immer-



sion foot" and is thought by some investigators to have a prominent role in further damage in the recovery phase of frostbite<sup>(21)</sup>. (This opinion has led to the widespread use of anticoagulants and vasodilators in the immediate post-freezing period.) It is most evident when tissues are in the temperature range of +5 degrees to +15 degrees Centigrade<sup>(15-17)</sup>.

In view of these experimental and theoretical considerations it appears that the time the involved tissue is frozen should be minimized and that once thawing begins there should be rapid rewarming to normal body temperature.

Once initial rewarming is accomplished, the physician is faced with the equally important clinical problem of preventing secondary changes such as fibrosis of intrinsic muscles, sludging of blood, thrombosis of vessels and irreparable changes in peripheral nerves. With this in mind we decided to use a potent tool heretofore not employed in this problem, ultra-high frequency sound. The major advantage of ultra sound over conventional diathermy is deep penetration of tissues.

Standard commercial ultrasonic equipment, then available in 1955-56, appeared to suit this purpose. Its utilization, effective in whirlpool or water bath, permitted penetration to all deep structures found in the extremities, including bone. We have felt that the condition of structures deep to skin is the determining factor in the eventual outcome of the frozen extremity. Assuming this to be so, it seemed logical for initial therapy, that treatment be directed to the injured, but still viable, deep structures, especially vessels and nerves, as well as bone and intrinsic musculature including tendon. Treatment then was directed, not to the changes so glaringly apparent in the envelope of skin, but to the more important contents. The preservation of these, the deep structures, provides the maximum results, since skin may readily be replaced by the indicated graft procedure at the proper later time.

Unfortunately, the rapidity of cooling of the injured part and the total duration of cold exposure are not usually within the control of the clinician in naturally occurring frostbite. In addition, most cases have already undergone thawing when seen first. (The early treatment of frostbite cases will be discussed in Part II of this paper.) However, in accordance with the above principles, the following program of treatment was planned and

applied whenever possible to the cases which are the subject of this paper.

### III. METHODS

When first seen the patient was evaluated for any deficiency of general body heat and efforts were directed at first instance to restoring this by the general application of heat externally and internally by ingestion of warm liquids. Parts still frozen or cold were then brought to body temperature by immersing in a warm water bath at temperatures from 42 to 48 degrees Centigrade (110 to 118 degrees F.). A whirlpool bath was usually used which provided more rapid heat transfer than a simple water bath. This was followed with very careful cleansing of the part with thorough but gentle scrubbing with a germicidal solution, benign to the tissues. In most cases this consisted of a hexachlorophene-containing detergent (pHisoHex<sup>®</sup>). Scrupulous care was taken to avoid trauma to the tissues and to avoid puncture of any blebs present. Following the achievement of as nearly an aseptic state as possible, the involved part was placed at rest on a sterile sheet and covered with a cradle over which a second sterile sheet was arranged to prevent contamination and unnecessary contact. No dressings of any kind were used except for small pledgets of sterile cotton inserted between the distal phalanges to avoid maceration.

Physiotherapy was instituted immediately when possible with whirlpool baths, usually combined with ultrasound therapy. Baths were given for twenty minutes once or twice daily for one to two weeks under as nearly sterile conditions as could be achieved. Active motion of the affected part was immediately encouraged. Passive manipulation was delayed until the acute stage had subsided and until danger of infection had diminished. Antibiotics were utilized in the same amounts and for the same rationale as are used with open fractures. Ultrasonic treatment was used in dosages of 1-1½ watts per square centimeter in a water medium, the sound head as close as possible without contacting the extremity. The application of this high frequency sound was prescribed for five minutes once or twice daily within the bath. In general, the method was directed towards the restoration of normal circulation, to the prevention of infection and ascending gangrenous change, and to the preservation and early rehabilitation of muscle and joint function.

(to be continued)

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# FROSTBITE:

## Experience with Rapid Rewarming and Ultrasonic Therapy

WILLIAM J. MILLS, Jr., M. D., ROBERT WHALEY, M.D., WINTHROP FISH, M.D.

ANCHORAGE

### PART II\*

In Part I of this paper, "Alaska Medicine," March, 1960, the general methods of treatment were described for those cases directly under our care. The background of knowledge concerning cold injury was briefly reviewed. In this portion of the paper, experience with fifty-one cases of frostbite is described. Of these, forty-one were treated directly by the senior author (WM) as either the attending or consulting physician. The remaining cases were drawn from the file of the Alaska Native Service Hospital or from Providence Hospital, Anchorage.

The majority of those patients not directly treated were followed by one of us as an interested observer. Only three of the total series were not seen by at least one of the authors. Hospital records were very carefully studied and any pertinent information was abstracted from them. The patients themselves were, if possible, interviewed at length concerning the cause of their exposure, the character of their exposure, and resulting symptomatology. All data which was considered of any possible value in the epidemiologic or clinical study of this injury was compiled. Only that information considered to be of general interest is presented here (Table I). Six cases of minimal to severe cold injury to the ears were not included in this series.

#### Group Characteristics

Table II outlines the racial and age distribution of the affected group. The majority of patients are, as one might expect, in the fifteen to forty-five years age range. It is of interest that Caucasian and Native patients are almost comparable in number. (Throughout this paper Native will be understood to refer to those of Aleut, Es-

\*These studies were aided by Contract Nonr-3183(00) (NR 105-249) between the Office of Naval Research, Department of the Navy, and William J. Mills, Jr., M.D.

TABLE II  
Age and Race Distribution

Age	Caucasian		Native	
	Male	Female	Male	Female
0-15	1	0	1	1
15-30	9	0	6	5
30-45	9	1	5	2
45-60	5	1	4	0
61-	1	0	0	0

kimo, or Indian extraction.) The appearance of only two cases of frostbite in Caucasian women is understandable in view of the infrequent exposure of this group.

In Table III the Caucasian and Native groups are listed according to cause of injury. The classification of "insidious" includes those cases of frostbite occurring inadvertently with the subject unaware of the severity of the cold exposure. This is as opposed to that group where exposure was appreciated but could not be avoided. A separate classification is given to include that group exposed as a result of alcoholic intoxication. They

TABLE III  
Cause of Injury by Race

Cause of Exposure	Caucasian	Native	T'tl
1. Insidious	1	3	4
2. Involuntary (Accidental)	18	14	32
a. Aircraft accident	3	2	
b. Land vehicle breakdown	3	0	
c. Hunting or trapping	5	4	
d. Injury at work	1	0	
e. Assault	2	2	
f. Runaway dog team	0	3	
g. Fell through ice in river	0	3	
h. Other	4	0	
3. Alcoholic Stupor	8	7	15
a. With assault		3	
Total	27	24	51



represent an individually large class and apparently a serious health hazard in this state.

Table III demonstrates some expected findings. These include a preponderance of Caucasian injuries in those exposed through truck or automobile breakdown or accident, and of Native injuries from runaway dog teams or from penetration of ice while crossing rivers or lakes covered by overflow. A number of unexpected findings occur, however, including the majority of insidious injuries revealed in Natives. This is somewhat contrary to the common conception that these people are highly experienced and therefore less liable to sustain cold injury of this type.

The assault group included those rendered helpless or unconscious by attack. Most of these were female, and most reported flight after the assault in sub-zero weather, inadequately clothed, with average exposure of from thirty minutes to two hours prior to obtaining aid or rewarming.

In all the above, exposure for most patients was at temperatures well below  $-20$  degrees Fahrenheit or, in a few cases, in the range of just above zero accompanied by high winds.

Classification of Injury

In attempting to evaluate the end results of the cold injury, a scheme of classification in roughly progressive degrees of severity was chosen as outlined in Table IV. Generally, the degree of restoration of function of the injured extremity should be considered the most important measure of result. However, although many of these patients have been followed for several years subsequent to their injury, the most recent sustained injury only one month prior to this writing. It is not possible to completely assess functional results at this time, since we were not able to see all of these patients for a proper follow-up. Too, the results are in some cases taken from descriptions rendered by other physicians, and these patients have not as yet been completely evaluated

TABLE IV	
Classification of Degree of Ultimate Injury	
A—No recorded or demonstrated residual	
B—Dysesthesia	
Intrinsic muscle atrophy	
Skin loss requiring replacement skin cover	
Limitation of joint motion greater than 25%	
C—Phalangeal amputation, any level, three or less	
D—Phalangeal amputation, any level, four or more	
Complete phalangeal loss at metacarpal or metatarsal—phalangeal junction	
Major amputation of the extremity	

in terms of our classification. To be objective, in measuring severity of injury, the classification is based entirely upon anatomical grounds.

The “A” Group includes only those patients without recorded or demonstrable residual. In those cases treated by the authors, no case of significant cold injury was found which did not have some residual, even though minor. Therefore, this group contains patients mainly treated by others. Group “B” contains those patients demonstrating any dysesthesia, or any sensory disturbance including hyperalgesia, hypesthesia, paresthesias, or causalgia. Also grouped in this “B” category are those cases demonstrating intrinsic muscle atrophy, skin loss requiring replacement, or limitation of joint motion greater than 25% of the normal range.

TABLE V				
Severity of Injury by Race				
	A	B	C	D
Caucasian	2	14	6	5
Native	2	12	6	4

Group “C” includes those patients who usually demonstrated all of the above but who sustained anatomical loss not exceeding three phalanges.

Group “D,” on the other hand, consists of those who had tissue loss greater than Group “C,” and in general represents those whose amputations are significantly handicapping. In the majority of cases, the Group “D” result amounted to the loss of a foot or lower leg, often bilateral (or separation at the metatarsal-phalangeal junction). Throughout the paper these specific groups of injury are used in evaluating the results of diverse methods of thawing and treatment. They represent our best measure of the treatment method and the subsequent end result of the injury.

Results

In Table V the net results, in accordance with the above classification, are tabulated for Caucasians and Natives. There is no significant difference between these two groups. It is seen that there are only four patients in Group “A.” These, as noted above, were not in the authors’ treatment series. Such patients so described by others, who were re-examined by us, were invariably found to demonstrate some residual limitation of motion or neurovascular loss. These were classified then in

Group "B." It is our conclusion that if actual tissue freezing occurs, even though the injury is relatively superficial, some demonstrable objective or subjective residual will persist indefinitely.

**TABLE VI**  
**Degree of Injury vs. Intoxication**

	A	B	C	D	Total
Intoxicated	1	8	4	3	16
Not Intoxicated	3	18	8	6	35

Because of the many loose statements and multiple theories regarding the effect of alcohol on frostbite, before and after exposure, Table VI has been included. There appears no significant difference in the distribution of intoxicated or non-intoxicated patients when classified by degree of injury. It is obvious from individual case histories that many of those who were intoxicated and sustained frostbite presumably would not have received this injury had they been aware of their environment and able to care for themselves.

In Table VII the subjects are classified according to anatomical areas of injury, race and final result. It is seen that the feet are involved more often than other sites, and are particularly likely to be involved bilaterally and often symmetrically. It is apparent from individual cases that involvement of the feet often occurs first,

**TABLE VII**  
**Anatomical Site of Injury**

Area of Injury	Cauc	Nat	Ttl	A	B	C	D
H, F, Bilat.	3	5	8	0	3	5	0
H, Bilat.	3	1	6	0	3	2	1
F, Bilat.	16	11	27	0	18	4	6
H, Unilat.	1	3	4	2	2	0	0
F, Unilat.	1	2	3	2	0	0	1
OTHER	1	2	3	0	1	1	1

followed by injury to the hands. This is the usual occurrence in those wearing the standard type of winter clothing, who sustain severe exposure with body cooling.

The preponderance of Caucasian subjects with involvement of both hands only is interesting. This appeared as a result either of accidental loss of the hand covering due to wind, of loss in the

course of the accident preceding the cold trauma, or of a lack of proper hand cover prior to cold injury. It is of further interest that the majority of major amputations of those subjects falling in Group "D" sustained foot injuries. Cold injury to the hand rarely produced more than isolated phalangeal loss. (Caucasian-Native racial variation, particularly regarding hand injuries, will be discussed in Part III.)

#### Variation of Result by Method of Treatment

The treatment of frostbite as advanced by the authors in Part I of this paper consisted of the rapid restitution of body temperature by warm oral liquid intake and a warm total bath when this was possible, which was seldom. This was followed immediately or concurrently by the rapid rewarming of the affected part by submersion in a water bath, 110 to 118 degrees Fahrenheit, preferably in a whirlpool. Treatment thereafter utilized whirlpool baths containing pHiso-hex® solution once or twice daily at body temperature. In some cases the use of ultrasound was combined with the whirlpool bath.

Initially antibiotics were utilized for the first two weeks of therapy. The choice of antibiotic varied. Our particular choice had been that of a combination of streptomycin and penicillin. In recent cases no antibiotics have been used in the treatment of these patients, unless the presence of infection was identified.

All attendants, particularly in the early stages, were required to wear clean gowns and face masks. In all our cases, the treatment of the injury was by the open method, without the use of occlusive dressings or ointments. It was found that some time after the fourteenth through the twenty-fourth day, with much variation in this time, sufficient drying of the tissues occurred so

**TABLE VIII**  
**Results by Type Hospital Treatment**  
**Authors' Method vs. All Other**

	A	B	C	D	Total
Full F.B.R. Program (R.R., U.S., W.P., P.T.)	0	6	1	0	7
U.S., W.P. & P.T.	1	4	2	1*	8
W.P. & P.T.	1	4	1	0	6
Total Above	2	14	4	1	21
Other Methods	2	12	8	8	30
* See Text					



that clean sheets or drapes might be substituted for the sterile cover.

The investigators had very little control of early treatment in most of the subjects available for study. Patients are categorized by the type of treatment received in Table VIII.

Seven patients received the full treatment as outlined. Six of these could later be classified in Group "B" and one in Group "C." The second group noted above consisted of eight patients who were allowed to warm by being placed in a warm room, but who were seen soon after, and received whirlpool, ultrasound and physical therapy. This resulted in one Group "A", four Group "B", two Group "C", and one Group "D".

Six patients received neither rapid rewarming nor ultrasound, but did receive the essentials of the program which consisted of open treatment with daily whirlpool and physical therapy, and this resulted in one Group "A", four Group "B", and one Group "C". The totals of these three groups are tabulated and compare in number with the total patients treated by other methods (thirty in number) wherein there was much greater anatomical loss. Of these, a greater percentage fell in Group "C" and "D" and this is a statistically significant difference.

The initial treatment of this series of patients varied widely depending upon where and by whom they were first seen. Of the total of fifty-one subjects, only seven were treated by "rapid rewarming." In many cases, of course, the extremity had thawed and risen to near body temperature by the time the patient reached trained medical aid. This occurred either during the period of rescue, transportation, or while awaiting examination or treatment in a warm room. One case\* thawed his own feet by exposing them to dry heat from a diesel generator exhaust. This case resulted in bilateral amputation of the tarsal-metatarsal junction. The tissue temperature is apparently readily raised to a lethal level when the injured tissue is not being perfused with blood.

Six subjects were thawed by immersing the foot in snow or ice water to achieve slow thawing (Table IX) in accordance with principles widely advocated in the past and by some authorities today. It is important to note that though this is a small series, half of these patients sustained major amputation, that is, fell in Group "D", and two-thirds, or four, of them sustained some anatomical loss. By comparison, of the patients treated by

"rapid rewarming," or of those allowed to thaw at room temperature, less than 10% sustained major amputation, and only approximately one-third demonstrated any tissue loss. Despite the small number of cases, this is in accordance with

**TABLE IX**  
**Result by Method of Initial Treatment**

	A	B	C	D
Rapid rewarming (water bath 110°-120° F)	0	6	1	0
"Thawing" with ice, snow or ice water	1	1	1	3
Other means, predominantly room temperature	3	19	10	5
Excessive dry heat	0	0	0	1

the general findings in animal experimentation. Although the proportion of those sustaining no anatomical loss in the group with "rapid rewarming" is considerably greater than that among those thawed at room temperature, the differences are not statistically significant in this small series.

Patients not treated directly by the authors received a variety of treatment. This included whirlpool and physical therapy in some cases, while others were treated with occlusive dressings, and a few with either anti-coagulants, vasodilators, or sympathetic block. None of the last were used on a sufficient number of patients to warrant statistical consideration.

Two major categories of treatment consisted of the "Open" as opposed to the "Closed" method. The "Open" method is defined as treatment using absolutely no cover, or dressing of any kind. The "Closed" method is descriptive of the utilization

**TABLE X**  
**Result by Whether Closed or Open Treatment Was  
Used—All Cases**

	A	B	C	D
Open method (no cover)	2	17	4	2
Closed method (occlusive dressing)	2	9	8	7

of occlusive dressings, with or without ointments or salves, moist dressings, or incorporation of the part in plaster of Paris. Twenty-five patients were treated with the "Open" method, and of these, six sustained anatomical loss (Table X). In this group were two major amputations. On the other hand, twenty-six patients were treated by



"Closed" methods, and of these fifteen had sustained anatomical loss with seven major amputations. Cause for this difference, which appears significant, is discussed later, but a portion of the

**TABLE XI**  
**Days Hospitalized by Method Treatment**

Days Hospitalized	Open	Closed
0- 10	3	5
11- 30	4	0
31- 60	8	6
61-120	7	5
121 or more	3	10

difference appears to be in the dissimilar incidence of infections utilizing the two methods.

Deep cellulitis appeared more frequent in the "Closed" method of treatment. This difference is

**Table XII**  
**Result by Presence or Absence of Infection, Any Severity**

	A	B	C	D
Without infection	3	18	1	2
With infection	1	8	11	7

reflected in the somewhat longer hospitalization of the severely injured patient (Table XI).

Important to the final result, as evidenced by this series of patients, was the presence or absence of infection (Table XII). For this separation, infection was considered to consist of any demonstrable evidence of purulent material, regardless of amount, and was, in most cases, demonstrated by bacteriological culture.

**TABLE XIII**  
**Result of Use of Debridement**

	A	B	C	D
Without debridement	3	20	0	3
With debridement	1	6	12	6

In Table XIII the subjects are tabulated without regard to other features of their treatment, initial or late, to demonstrate the relationship of debridement to result. The distribution is seen to correspond closely to the previous table, Table XII. Debridement was considered to mean intervention surgically, by isolated or periodic removal of superficial tissues with any instrument, but not

amputation. Rupture of the blebs mechanically was considered debridement.

In order to determine the features responsible for this apparent relationship, the subjects were again re-classified (Table XIV). This classification was arranged to distinguish between those in

**Table XIV**  
**Debridement and Infection vs. Result**

	A	B	C	D
Not debrided and not infected	3	16	0	2
Not debrided and infected	0	4	0	1
Debrided and not infected	0	2	2	0
Debrided and infected prior to debridement	1	3	2	1
Debrided, without prior infection and infected subsequent to debridement	0	1	8	5

which debridement was performed because of severe infection and those in which infection was not prominent at the time of debridement but followed the procedure. We feel that this tabulation is important.

It will be seen that good results generally were obtained in those patients who (1) were neither infected nor debrided, (2) were infected but not debrided, (3) were debrided but not infected. On the other hand, poor results were obtained in those patients who were infected and debrided, or in a fairly large group of patients who were debrided and developed infection following debridement. It should be noted here that of twenty-six patients of all types not treated by debridement, only three sustained significant tissue loss. Of seventeen individuals not infected at the time of debridement, fourteen, or all but three, suffered significant tissue loss and five sustained major amputation.

We consider it overwhelmingly demonstrated here that of all the factors in the treatment of frostbite which may influence the result, premature surgical intervention by any means, in any amount, is by far the greatest contributor to a poor result of any variable analyzed.

An attempt was made to evaluate the method utilized by the authors in regard to the number of infections sustained during the course of treatment. For this purpose, the series was divided into three categories, to include those treated by our methods, those treated by other methods but utilizing whirlpool bath, and those utilizing any treatment excluding whirlpool.

Initially the patients were classified as demonstrating the absence or presence of infection. When so presented, this series of subjects demonstrated almost 50% infection regardless of treatment. However, when re-classified into superficial or deep, a marked difference appeared. This classification was not an arbitrary one, since those classified as superficial consisted of small, infected pockets, or pustule formation in the eschar or

final estimate as measured by tissue loss. Superficial injury was defined to be injury limited to skin, corresponding to "first" and "second" degree injury of many writers. Deep injury involves tissues below the skin including muscle, tendons, nerves, blood vessels and bone. This is comparable to "third" and "fourth" degree injury. It is apparent from a glance at the figures that there is little correlation between the initial estimate of injury and the final result.

**TABLE XV**  
**Infection by Method of Treatment**

	No Clinical Infection	Superficial Infection	Deep Infection incl. Osteomyelitis
F.B.R. Program	11	10	0
Other methods with whirlpool	3	5	0
Other methods without whirlpool	10	3	9

tissues adjoining it. Deep infections, however, invariably consisted of extensive cellulitis, with or without osteomyelitis. No patients were seen who fell between these extremes.

Results of this distribution are shown in Table XV. It will be seen that although both our recommended program and other methods which included whirlpool had a significant number of infections, these were entirely superficial. Other methods, however, without the use of whirlpool therapy, developed infection that quite often progressed to become deep, and usually resulted in serious tissue loss. Our clinical impression has

**TABLE XVI**  
**Original Estimate of Injury vs. Result**

	Result	
	Superficial	Deep
Original Estimate		
Superficial	21	17
Deep	4	9

been that daily whirlpool therapy with antiseptic cleansing solution is an effective method to prevent the development of a serious infection, and we feel this is confirmed by these results.

Table XVI represents our subjects, classified according to the original estimate of degree of injury, superficial or deep, plotted against the

Most of the original estimates of the degree of injury were made by one of us (WM) who had considerable experience with frostbite. It is apparent that the judgment of the severity of injury soon after its occurrence is extremely difficult if not impossible. The further classification of frostbite, particularly in the initial stages, into "degrees" such as has been attempted by many authors, must necessarily be tentative, and unreliable. Further, it has no clinical usefulness apparent to us. We have accumulated no data that would indicate a variety of treatment methods dependent upon the diagnosis of depth of frostbite.

In Table XVII, the salient features of those patients falling in Group "D" are summarized. It is interesting that five of the patients had debridement followed by infection, and of this group one patient sustained bilateral major amputation prior to the fourteenth day. His course subsequently was followed by soft tissue infection at the amputation site, and osteomyelitis of the distal portion of the amputation stump. He required revision of both amputation sites due to persistent infection.

Two of these patients, involved in an airplane accident, sustained such injury, that amputation was performed in the absence of debridement or infection. In one, the vascular trauma was so great that all pedal pulses in the foot were absent and examination upon initial admission demonstrated a fracture dislocation of the ankle and the navicular, the latter remaining unreduced. As a lifesaving measure, and in order to obtain help for his companion, he had crawled with the above noted injury over eight hours, at least three and one-half miles down a mountainside in sub-zero weather in the Arctic. It is logical to assume that he sustained considerable vascular injury during this travel. His injuries included a fracture of the lumbar spine as well. In this particular case, spontaneous separation of the tissues occurred at the tarsal-metatarsal junction.



**TABLE XVII**  
**Features of Those Cases with Major Amputations**

Case	Area	Age	Race	Assoc. Injury or Condition	Factors Preceding Amputation	Level of Amputation
10	Fr-l	17	E		Severe penetrating deep cold injury. Early demarcation, dry gangrene, left distal foot. Debridement followed by infection and retraction of tissues.	Trans-metatarsal left foot
18	Fl	41	E	Fx lumbar spine Fx-dislocation left ankle Dislocation left navicular	Vascular trauma incident to crawling on hands and feet 3-4 miles, 8 hrs. dragging L leg. Unreduced navicular dislocation.	Middle third left tibia. (Preceded by spontaneous separation tarsal-metatarsal junction)
22	Hr, Fr	36	E	Fx Metacarpal R hand Fx tibia (closed) R Fx talus, R	Cold injury superimposed upon multiple fx's, R lower leg. Vascular deficit.	Mid third right tibia.
23	Fr-l, Hr	54	E	Starvation Dehydration	Deep penetrating cold injury. Thawing with ice and snow water.	Bilateral metatarsal phalangeal junction (level of demarcation)
31	Fr-l	42	C	Alcoholic stupor	Debridement, infection, early amputation prior to 14th day, osteomyelitis	1) Proximal 3rd left tibia 2) Tarsal-metatarsal junction right
33	Fr-l	42	C	Alcoholic stupor	Multiple episodes "frostbite", cold exposure after alcoholic bout. Superficial infection, debridement, osteomyelitis and multiple phalangeal amputations	Complete bilateral phalangeal loss, all toes, L&R
38	Hr-l	69	C	Alcoholism Organic brain syndrome	Thawing, ice packs, early debridement, infection.	Phalangeal amputations bilateral, excluding thumbs.
46	Fr-l	46	C	Alcoholic stupor	Early debridement, early surgical procedures (grafts) infection, osteomyelitis	Bilateral proximal third tibial amputations
49	Fr-l	28	C		Severe penetrating cold injury. Thawed at excessive dry heat 165-185° F. Rapid deep demarcation, tarsal-met. junction.	Bilateral tarsal-metatarsal junction

Amputation was performed at the mid-tibial level on a further patient, with such skeletal injury of tibia and talus, including interarticular injury, to preclude, in the opinion of the surgeon caring for him, a good terminal result. One other patient, whose extremities were thawed with ice and snow water, received such deep penetrating cold injury, and was without treatment for such a length of time, that spontaneous demarcation occurred. His loss was bilateral at the metatarsal-phalangeal junction. Another patient, with thawing by ice packs, had an unfortunate result involving both hands, with associated early debridement and infection.

The patient previously referred to, who thawed his feet in a diesel generator exhaust, underwent rapid demarcation with bilateral loss at the mid-foot. Despite excessive warming, he sustained, with the open method of following treatment and constant whirlpool therapy, no evidence

of deep infection. After multiple skin grafts, he obtained a good functional result and required no further amputation.

#### Ultrasound

We have been interested in the utilization of ultrasound as a penetrating tool and an adjunct therapy in the treatment of frostbite. It has been used in these cases only in conjunction with whirlpool. The results of ultrasound at this time are inconclusive. Only recently have we attempted its controlled unilateral use in cases with bilateral symmetrical injury of hands and feet. An insufficient number of such control cases have been accumulated to draw valid conclusions (Table VIII). In this table eight patients received therapy, utilizing ultrasound, whirlpool and physiotherapy without "rapid rewarming."

Six others were treated very similarly, excluding the use of ultrasound. No marked difference is seen.



TABLE I  
SUMMARY OF 51 PATIENTS STUDIED

Pt	Sex	Age	Race	Cause of Exposure	Complicating Factors	Anat. Area	Method of Thawing	Method of Treatment*	Imp	Deb	Infection	Group†
								RR US WP PT O C				
1	M	24	E	Insidious	Ment. deficiency	Fr-l	Delayed: Room Temp.	X X	X	S	Superficial	B
2	F	16	E	Flight after assault	Inac. Pulmon. Tbc Gen. hypothermia	Fr-l, Legs r-l	" "	X X	X	D	None	B
3	M	46	I	Alcohol	Gen. hypothermia	Hr-l	" "	X	X	D	None	B
4	M	21	E	Alcohol	Urinary tract Tbc (act.)	Fr-l	" "	X X X	X	S	Superficial	C
5	M	31	E	Insidious Ice Fishing		Fl	" "	X X X	X	S	None	A
6	F	8	I	Accident	Gen. hypothermia	Fr-l	" "	X	X	S	None	B
7	M	51	E	Alcohol		Hl	" "	X X	X	S	Superficial	A
8	F	27	E	Alcohol		Fr-l	" "	X	X	S	Deep	C
9	M	19	A	Fishing		Hr-l, Fr-l	" "	X	X	S	Deep	C
10	F	17	E	Fell through ice crossing river		Fr-l	Thawing: Ice & Snow	X X X	X	D	Superficial	D
11	M	50	I	Fell through ice crossing river	Pulmonary Tbc active	Fr-l	Delayed: Room Temp.	X X X	X	S	None	B
12	M	23	A	Insidious	Old laceration med. mn left hand	Hl	" "	X X	X	S	Superficial	B
13	M	42	I	Lost in blizzard, runaway dog team	2nd episode F.B. (15 yrs ago) blind- ness, cong(?) Gen. hypothermia	Hr-l, Fr-l	Rapid Rewarming Water Bath-Tub 118° F	X X X	X	D	Superficial	C
14	F	18	E	Fled in bare feet Assault	Sever Rh Ht disease with A.I.	Fl, Leg r	Rapid Rewarming Hot packs	X X X	X	D	None	B
15	F	23	I	Alcohol-Assault	Gen. Hypothermia	Buttocks, thighs	Delayed: Room Temp.	X	X	S	Superficial	B
16	M	15	E	Fell through ice crossing river	2nd episode F.B. Pulmonary Tbc act.	Hr-l, Fr-l	Thawing: Ice & Snow	X X X	X	S	Superficial	B
17	M	29	E	Unclothed in snow	Schizophrenia, org. brain syndrome	Fr-l	Delayed: Room Temp.	X	X	D	None	B
18	M	41	E	Aircraft accident	Fx lumbar spine Fx dislocation left ankle, dislocation left navicular	Fl, Leg l	" "	X X	X	D	None	D
19	F	32	E	Flight from assault - Alcohol	Starvation dehydration	Fr-l	" "	X X X	X	S	None	B
20	M	35	E	Runaway dog team		Fr-l	Thawing Snow: H.O	X X X	X	D	Superficial	C
21	F	44	E	Flight from assault - Alcohol	Inact. pulmonary Tbc	Fr-l	Rapid Rewarming Hot packs	X X X	X	S	None	B
22	M	36	E	Aircraft accident	Fx right tibia Fx right talus	Hr, Fr, Leg r	Delayed: Room Temp.	X	X	D	None	D
23	M	54	E	Runaway dog team	Starvation Gen. hypothermia	Hr-l, Fr-l	Thawing Ice Packs, Snow	X X X	X	D	Superficial	D
24	M	26	E	Trapping in snow storm		Hr-l, Fr-l, Ears	Delayed: Room Temp.	X	X	D	Deep	C

25	M	22	C	Vehicle accident	Loss of gloves and shoe packs	Hr-l, Fr-l, Ears	Rapid Rewarming (110-115°) Bath	X	X	X	X	D	L	Superficial	B
26	M	30	C	Vehicle breakdown	Gen. hypothermia mentally confused	Hr-l, Face	Rapid Rewarming Hot Baths	X	X	X	X	S		Superficial	B
27	M	42	C	Injury on trail	Fx left ankle Bimalleolar	Fr-l	Rapid Rewarming WP (118° F)	X	X	X	X	S		None	B
28	M	16	C	Hunting accident	Open Fx left tibia Crawled 3 mi. to aid Gen. hypothermia	Fr-l, Knees, Legs	Delayed: Room Temp.		X			S		None	B
29	M	29	C	Insidious	History of old periph. vascul. dis.	Fr-l	Rapid Rewarming (Soaks - 115° F)	X	X	X	X	S		None	B
30	M	8	C	Exposure after assault	Gen. hypothermia	Hr-l, Fr-l	Delayed: Room Temp.	X	X			S	E	Superficial	C
31	M	42	C	Alcohol	Gen. hypothermia	Fr-l, Legs	"					S	E	Deep	D
32	M	36	C	Found in snow alcohol	Gen. hypothermia	Hr-l	"		X			S	L	Deep	C
33	M	42	C	Found in snow alcohol	Gen. hypothermia	Fr-l	"			X		S	L	Deep	D
34	M	29	C	Hunting accident	Mentally confused	Fr-l	"	X	X	X		S		None	B
35	M	44	C	Bromide intox. alcohol		Fr-l	"	X	X	X		S		None	B
36	M	36	C	Alcohol	Hallucinating	Fr-l	"	X	X	X		S	E	Superficial	C
37	M	41	C	Aircraft accident	Post injury psychosis	Hr, Fr-l	"	X	X			S	E	None	C
38	M	69	C	Found exposed in snow, head injury	Chronic alcoholism	Hr-l, Knees	Thawed ice packs				X	S	E	Deep	D
39	M	32	C	Aircraft accident	Sprain left angle	Fr-l	Delayed: Room Temp.	X	X			S	E	Superficial	B
40	M	27	C	Aircraft accident	Fx ulner, right	Fr-l	"	X	X			S	E	None	B
41	M	53	C	Trapping		Fr-l	"			X	X	D	E	Superficial	C
42	F	51	C	Exposure, not properly clothed		Fr-l	"			X		S		None	B
43	M	48	C	Changing truck tire		Hr	"			X		S		None	A
44	M	23	C	Hunting		Fr	Ice packs				X	S		None	A
45	M	26	C	Hunting		Fr-l	Delayed: Room Temp.		X			S		Superficial	B
46	M	46	C	Alcohol		Fr-l	"			X		S	E	Deep	D
47	M		C	Mt. climbing accident		Hr-l	"			X		S	E	Deep	C
48	M	51	C	Mt. climbing accident	Diastasis (T-F) left Sprain right ankle	Hr-l, Fr-l	"			X		S		None	B
49	M	28	C	Vehicle breakdown		Fr-l	(185° F) Excessive Dry Heat	X	X			D	E	Superficial	D
50	F	30	C	Alcohol		Fr-l	Delayed: Room Temp.	X	X	X		S		None	B
51	M	53	C	Alcohol		Hr-l	"	X				S		None	B

\*RR—Rapid rewarming

Imp—Initial impression of severity of injury, superficial or deep;

US—Ultrasound;

†See Table IV

WP—Whirlpool;

PT—Physical therapy;

O—Open method;

C—Closed dressing;

Deb—Debridement.

# FROSTBITE:

## Experience with Rapid Rewarming and Ultrasonic Therapy

WILLIAM J. MILLS, Jr., M.D., ROBERT WHALEY, M.D., WINTHROP FISH, M.D.

ANCHORAGE

### PART III (See Dec., 1960)

From review of the data in Part II†, it is apparent that the elimination of freezing injury is improbable. A decrease in incidence is possible, with proper prophylaxis. General knowledge of proper clothing, especially hand and foot gear, and care and use of such gear when in freezing temperatures is essential. Basic information regarding the mechanism of heat production and loss, resulting from the effect of activity, fatigue, sweating, shivering, wind exposure, and wet clothing, should be made available to winter travelers in the Alaskan area.

Methods of care in the event of unavoidable trauma to the person should be taught those whose occupation or avocation permits exposure in the Arctic or sub-Arctic. Neither here nor elsewhere has a solution been found nullifying the effect of neglect, carelessness, alcoholic intoxication, accident or assault that were contributing causes in this series.

Regardless of cause, it is interesting that only six of our fifty-one subjects were persons not oriented or acclimatized to Arctic or sub-Arctic conditions. Most of our severe injuries occurred in lifetime or longtime residents of Alaska.

The effect of possible training and conditioning on resistance to cold injury is still a highly controversial subject. In this respect, the relative paucity of cases of hand injuries among Native peoples here in Alaska is striking. Longtime Caucasian residents in the Alaska wilderness, as well as the Native people, traditionally utilize the hands fishing and working in the cold to a greater extent than urban dwellers and military personnel.

The construction of Native fur clothing is such that heat control of the body is quite difficult by removing or adding clothing. Temperature regulation often is accomplished in the Eskimo and Indian, by the removal of gloves, even in sub-zero weather, and the utilization of face and hand circulation as a heat radiator. The work of Irving, Nelms, and Ellsner<sup>(1)</sup> has demonstrated the greater ability of the Arctic Indian to perfuse his hands when subjected to cold exposure. He is able to achieve hand vasodilation more rapidly after cold stress, to achieve a much higher flow rate, and to sustain it despite general body chilling, in differences that are marked over the normal white control subject.

The difference is such that these people are able to work continuously for long periods with hands in water at freezing temperatures during their winter fishing or other activity, without obvious incapacity. Whites living a similar existence demonstrate an ability in this respect intermediate between the two groups mentioned. It is worthy of note that the Eskimo does not acclimatize his foot circulation in similar fashion.

In many patients the onset of freezing was apparent and gradual. A number of patients were aware of the danger besetting them, but were forced to choose between extremity freezing and survival. In some with associated injury to head, trunk or limb, no choice was available. Only great determination to survive, or opportune discovery, prevented eventual death from general hypothermia.

The initial treatment of freezing injury, as in any trauma, is one of first aid. Superficial skin involvement may be treated by warming the frozen part (especially head and face area) with a warm hand. Feet and hands may be warmed by contact with skin under protective clothing, and gloves and footgear may be changed or dried. Shelter, if available, resolves many of these problems.

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In cases of severe frostbite occurring on the trail, it is our impression at present, that the limb may best be left in its frozen state until rescue is performed, or shelter obtained. At that time, more definitive care, particularly rapid rewarming in a water bath, may be offered. There is good cause to feel this is proper.



*Fluff dressing under adhesive gauze, for transport. Blebs still intact after 24 hours of travel.*

Travel may be possible on a frozen foot, or feet, that remain so, and survival may depend upon this. After thawing, travel is difficult, if not impossible, on the extremity that is swollen, painful, hyperemic and blistered. If further travel is necessary, after thawing, in the same freezing environment, the danger of re-freezing is likely, and seldom is the result other than disastrous. Severe tissue loss in a recent Mt. McKinley climber, not included in this series, appeared due to a freezing injury, following by thawing, and then on further ascent, re-freezing.

Inspection of Table I of this paper will demonstrate that the total duration of freezing is not correlated with the final degree of injury, and may indicate that the final result is less a function of duration of freezing and more a function of method of thawing, particularly rapidity of thawing, or rewarming.

Our data is not conclusive on this point, but highly suggestive, and as more cases accumulate, this problem will be given much attention. It appears that little is lost by permitting the extremity to remain frozen or cold, if rewarming may be anticipated in the reasonable future.

## Thawing Methods

Our data seems clearly to indicate that the use of dry heat is hazardous because of the danger of raising tissue temperatures above the limit of viability, especially tissues previously traumatized by excessive cold. Aside from this, superficial injuries can be treated by most any method with apparently reasonable good results. At least no great difference in result was demonstrated in this small series, other than a definite earlier return to normal sensation and appearance, in the extremities rapidly rewarmed in a water bath.

Following deep injury, our best results were obtained in those cases rewarmed in a water bath, as described. This method is more painful in its initial stage, resulting in increased hyperemia and larger blebs or bullae. The pedal pulses often are "pounding," after warming, and the blebs usually extend distalward to include even the terminal phalanges. Because of the extreme reaction, particularly the edema and bleb formation, this is not a "trail therapy."

Although results from 'gradual thawing' or thawing at room temperature in superficial injury appear not different statistically from other methods, there is obvious evidence clinically in the 'deep injury' cases, that this method yields poor results, often associated with considerable tissue loss.

The treatment of deep frostbite by ice, ice water immersion, or snow, plainly results, even



*Deep freezing injury, 24 hrs, post rapid rewarming, early bleb development.*

in this small series, in greater tissue loss. It appears to have little theoretical or experimental basis to recommend it. It is likely, we feel, that this traditional method arose from, first, the lessened pain and discomfort in this method of thawing, particularly in superficial injury, and from obviously disastrous results of the application of external dry heat.

This effect of excessive dry heat probably accounted for the severe gangrenous results reported by Larrey during Napoleon's retreat from



*Effect of excessive dry heat, 98 days post injury.*

Moscow, a report so often quoted in the cold injury literature, that rewarming at temperatures greater than body temperature was for many years rejected, and only recently received encouragement from the published work of Aryev<sup>(2)</sup>, Crismon and Fuhrman<sup>(3)</sup> and Meryman<sup>(4)</sup>.

### General Management

Prior to thawing, transport of the individual or handling of the frozen extremity is ordinarily not a problem. The limb itself is splinted by cold and requires only that tight gear be loosened. If thawing has occurred, the part may be covered with loose fluffs, surrounded by adhesive gauze, or elastic dressings to prevent bleb rupture. If none of these dressings are available, then transport is difficult. The friable, edematous tissues,

covered often with blebs, may be injured during travel. Maceration and trauma to these tissues increases the risk of infection.

After recovery from general hypothermia, if present, and rewarming of the part, our patients began the previously described frostbite regimen. The initial examination is particularly important in the alcoholic, irrational, and often hypothermic patient. This patient is most difficult to manage, especially prior to return to the homeothermic state.

Management of the accident victim may become a problem in therapy, since frostbite often is secondary to dislocation or fracture or crush injury of the extremity. These cases present combined technical difficulties other than the method of rewarming and frostbite care, and as a group are desiring of more study. Treatment here is particularly directed to preservation of the peripheral vascular system.

Dislocations or fractures embarrassing circulation by pressure must be reduced and placed in such alignment to prevent ischemia. Traction increasing vasospasm is obviously contraindicated. Open reduction in an area of frostbite is hazardous.

After placement of the cradle for the lower extremities or sterile sheets for the upper extremities, the patient is equally dependent for a good result upon capable and understanding nursing care. This is especially so when the gangrenous changes are advanced. The frostbitten extremity is not "pretty" and often repels both nurse and patient. Very cold or very warm liquids should not be in contact with the insensitive extremity, in order to avoid further tissue embarrassment. Extremes of heat or cold may precipitate further gangrenous change. Control of infection, so ready to occur in these tissues, demands aseptic nursing technique. Clean gowns and masks are essential in the early edematous, hyperemic stages.

The irrational, alcoholic, or belligerent patient must be prevented from weight bearing, causing maceration of blebs and tissue contamination. In the event of absolute inability to control such a patient, and in one where techniques of restraint might be most difficult because of the involvement of the areas where restraints must be placed, we have found an Unna paste boot or even a light plaster cast over fluff dressings to be satisfactory



in the early stages. As soon as the patient is rational, these must be removed to permit a continuation of physiotherapy. Smoking is discouraged under this program.

A bright, cheerful hospital environment, ward area, or private room, is essential. Attendants and nurses must radiate confidence and render support. If deep or superficial gangrenous changes occur, with or without the associated odor, it is even more important not to set these patients away by themselves where they are unlikely to receive a normal degree of ward care and consideration. In fact, they require more care than most patients. Gentle handling of the extremities is necessary to prevent premature bleb rupture and tissue trauma or contamination.

We have found little promise in the use of vasodilators, anticoagulents, sympathetic block or sympathectomy in the early acute frostbite cases. Our own experience, however, is limited. Narcotic drugs have posed a considerable problem in the past. These patients often become dependent upon such drugs, even dependent upon the use of tranquilizing drugs. Few of them require sedation or analgesia after the first seventy-two hours. Much of their discomfort is the result of fear and concern. Most analgesic or narcotic drug therapy can be replaced by an encouraging bedside manner and a running explanation of the medical problem on daily rounds. It is essential that the attending physician and the nurses be patient and realize that, with adequate treatment and care, particularly after rapid rewarming, results will generally be much better than expected. As necrosis develops, and color changes become more marked, it is important to know that in all probability, fine epithelial tissues are growing below this superficial eschar. Little is lost at this point in delaying definitive procedures of any kind that are destructive of tissue. Often a pleasant surprise awaits both physician and patient when debridement is delayed or avoided, and physiological healing permitted.

### **Whirlpool. Physiotherapy and Ultrasound**

Physiotherapy serves at least two purposes. First, its active use, quite important from a functional viewpoint, permits patient participation in the therapy. The patient may leave his bed for definitive care, thereby altering his environment. At least once a day travel to the therapy area may well be a considerable morale boost, particularly in the case of bedridden patients or those hos-

pitalized for 30 - 150 days. The patient, too, is able to measure his progress by gradual increase in range of motion of the digits and the return of more normal sensation. Secondly, the use of whirlpool, with Hexachlorophene<sup>(R)</sup> detergent, cleanses the part of surface bacteria, and performs a physiological atraumatic debridement as well as permitting a stimulating massage of tissues. The difference in prevalence of severe infection in our series between those who did and did not receive whirlpool treatments is striking. There is little odor that so often is troublesome to these patients, following the use of whirlpool as compared with the patient who did not have this form of therapy. Whirlpool, too, possibly increases local circulation. It has, in our cases, appeared to cause rapid diminution of edema.

Pain is readily relieved after serial whirlpool. Patients, regardless of rewarming methods, tend to exercise the extremities while in whirlpool more than while lying in bed. Hand exercises, especially after the blebs have dried and sterile fluffs are utilized for pressure exercises (squeezing), are made easier and are more likely to be performed regularly during and after whirlpool therapy.

We have found ultrasound to be both help and hindrance. In cases of superficial injury, there is subjective evidence of benefit. Some patients report "improved" sensation and ease of interphalangeal joint motion. In several, assuming the cold injury to be nearly symmetrical in both extremities, with only one receiving ultrasound, there appeared a measureable change. Increased interphalangeal joint motion, and increased sensory return were marked. There was observed visual evidence of an apparent increase in vascularity of fingers and toes, particularly noted immediately after the use of ultrasound. With deep injury, or in the type of C and D results, there is cause to believe, from the sensation reported (boring pain, or dull ache), and from the appearance (hastened necrosis in some), that the use of ultrasound is harmful.

In some cases, after ultrasound, particularly those thawed by ice and snow and in a few, slowly warmed at room temperature, dry gangrenous tissues soon became wet. Liquifaction of the distal tissues was hastened in these. In further control groups, of approximate equal injury to each extremity, ultrasound has demonstrated its ability to cause resolution of the blebs many days



ahead of the opposite untreated extremity. At the third or fourth week, however, no difference was generally exhibited between those extremities.

### Debridement

From this study it is apparent that there exists no more traumatic therapy in cold injury than debridement. Mechanical rupture of the blebs converts the sterile field into a contaminated, potentially infected area. Incision of edematous and friable tissue permits ready access for bacteria, usually already present on the skin. From the gangrenous eschar, dry and wet, we have cultured in almost all our patients, staphylococcus aureus coagulase positive. The organism is provided a fertile culture media in these necrotic tissues. Too, incisions in these tissues, if carried too deep, will break through granulating epithelializing tissue below, and may result in further suppuration. Further, debridement or amputation in the early stages (first to third week) will penetrate the edematous tissues that then retract. More bone and joint surface is then exposed with further tissue loss.



*Premature debridement through wet, edematous tissues (52 days post injury) resulting in retraction of tissues.*

If amputation must be performed, it seems more proper to allow contracture and shrinkage of the tissues, with formation of a stabilizing granulation bed below at the demarcation site prior to the surgical procedure.

There may exist one area where debridement is beneficial if carefully performed. When the dry eschar in the resolving injury retracts and tightens, tissue necrosis below, or intrinsic muscle atrophy may develop. A fusiform digit or even loss of much of the digit may follow. If the eschar prohibits distal or proximal interphalangeal joint motion, a linear, volar or dorsal slit, even a bilateral digital slit may permit increased motion. This will provide, by increasing the range of motion, early shedding of the eschar, especially during whirlpool therapy.

Care in making these incisions must be utilized because of the underlying thin layer of new granulation tissue that is so readily penetrated and injured.

Debridement is adequately and physiologically performed by the whirlpool itself. The bath's gentle motion removes eschar as it is released from the epithelializing bed, only when it is physiologically prepared to separate.

### Infection

Superficial infection was present in most of our cases. Many demonstrated pockets of purulent material in the eschar. The superficial infection appeared held in abeyance by whirlpool, while increasing in many cases after debridement and without whirlpool.

For the first four years our patients were placed routinely on broad spectrum antibiotics. In the past year antibiotics were utilized only after definite indication and following culture and sensitivity studies. Their use was not essential in most patients utilizing whirlpool bath, but was more often required in patients treated otherwise.

Infection was found more often in those treated with occlusive dressings, not receiving whirlpool bath or daily lavage. It is presumed that the effectiveness of Hexachlorophene<sup>(R)</sup> in the bath was that of a germicidal agent, permitting constant topical application and was effective as prescribed because of its known property of reducing numbers of bacteria and interference with metabolism of pathogens as well as non-pathogens. It was used, too, as a prophylactic agent to decrease the incidence and severity of pyogenic skin infection. In our experience, only constant whirlpool followed by dry "open" technique will prevent the rapid growth of bacteria, particularly from the pockets of bacteria discovered on the gangrenous skin surface or the subcutaneous pools beneath the eschar.

## Amputation

The early appearance of the deep frozen extremity, once blebs have dried, is often that of a black mummified part, extending even above the digits and involving all of the extremity's surface. The overall appearance is often misleading. Amputation or debriding procedures at this period (6-21 days) may reveal viable deep structures, that would have permitted epithelialization under the eschar. Avoidance of premature amputation is essential. The eschar, after epithelialization, will often loosen and shed, permitting exposure of healing tissues below. If amputation must be performed, at the digital level, most satisfactory results appear to be at the sixty to ninety day period. The digits will, as all have seen, demarcate spontaneously, requiring only revision later. It is usually sufficient to remove the necrotic bone back to bleeding cortex or medullary canal, and permit the skin flap to fall over bone, utilizing then a small vaseline pack for three or four days. This may be held in place with one loose wire suture. Within seven days or even less, granulation has usually occurred that will permit further whirlpool therapy.



*Automatic debridement effect of whirlpool bath, with physiological separation of tissues.*

If, because of ascending infection, amputation must be performed at a higher level, it is absolutely essential to perform a strict Guillotine procedure. After edema has subsided and an adequate granular bed developed, revision of the stump can follow. All amputations were performed at extreme distal levels, to preserve as much tissue as possible. It is interesting to note that only one patient sustained amputation following rapid rewarming as described, and that resulted in loss only of a large toe.

## Associated Injury

Our experience with injury followed by secondary frostbite is similar to others. All cases of head, thoracic, abdominal or extremity injury must receive proper care, including restoration of



*Wet gangrenous change, thawed at room temperature (26 days post injury).*

general body heat and restoration of blood volume. Relief, if at all possible, must be provided the neurovascular deficit as early as it is recognized. The approach here is similar to that of an open fracture, where consideration is given first to preservation of life and limb, then function, and last of all, cosmetic result. Despite the glibness of this approach the problem is not that simple. Much clinical investigation is required to further the treatment of fracture dislocation or soft tissue injury of the extremity with associated cold injury.

Excluding patients whose psychotic reaction caused them to sustain cold injury as a result of deliberate exposure, or involuntary exposure unclothed, we are interested in the patient who becomes irrational or confused on the trail after freezing injury, and the bed patient who, after five or six days, demonstrates a severe emotional reaction, even developing hallucinations.

Langdon<sup>5</sup>, interested in this problem, has described this reaction as a response in some to "a situational reaction of adult life, usually in indi-



viduals not prepared for the eventuality of frostbite." This reaction has followed the traumatic occurrence, usually in a remote, forbidding area, associated often with considerable danger, and fear of failure of survival. In many, a hypomanic reaction has occurred. Few of these patients required psychiatric care of any prolonged nature.

They did require understanding, tolerance of their mood, patience, and constant visitation and encouragement. It appeared helpful to discuss the changes that were occurring to their extremities. Those that thus became a part of the program (and they are an active participant) progressed



*Exercise prognosis, against sterile fluffs, 36 days post injury, follow up Fig. A.*

more rapidly than others. One of our most reactive patients had considerable relief (and added much to our project) by using a recorder at frequent intervals at the bedside. Here he related, giving date and hour, changes in sensation and motion, or reaction to therapy, as these changes occurred. He gave us our best documented history of the occurrence of the injury, and discussed his reaction from initial onset through hospitalization to discharge. He was, upon release from the hospital, quite "expert" in cold injury, at least as involved his own episode.

Since it is important that the program be carried on without technique breaks, or premature ambulation, it has been found effective to have at

least two physicians or a physician and nurse, in some accord, follow these patients. One to demand strict adherence to principles of treatment, and the other a buffer to explain the need for this discipline and to offer sympathy if the treatment becomes too demanding and rigid. This problem becomes obvious when one considers the helplessness of the patient with freezing injury of both hands and both feet (eight of our series) who for weeks and months are unable to bathe, feed themselves, grasp objects, or attend to any personal needs, including bowel or bladder function, without second party support. They are, as noted by Langdon, essentially quadriplegic because of injury to all four extremities, and may have episodes of hypomanic behavior together with outright fear, frequent irritability, and occasional depression.

### Enzyme Studies

We have obtained standard enzyme determinations in our patients for the past two years. This portion of our project developed through an effort to demonstrate a pattern of vascular change in tissue trauma and repair during the period of re-warming and post re-warming. Too, we had originally proposed to seek a method utilizing alkaline phosphatase, lactic-dehydrogenase, serum glutamic oxaloacetic transaminase, and aldolase, as well as other standard enzymes, that would possibly provide a prognostic tool for early determination of definitive injury. This was primarily planned to provide the military field commander and medical personnel a tool for estimating disability in terms of availability for duty or priority for evacuation.

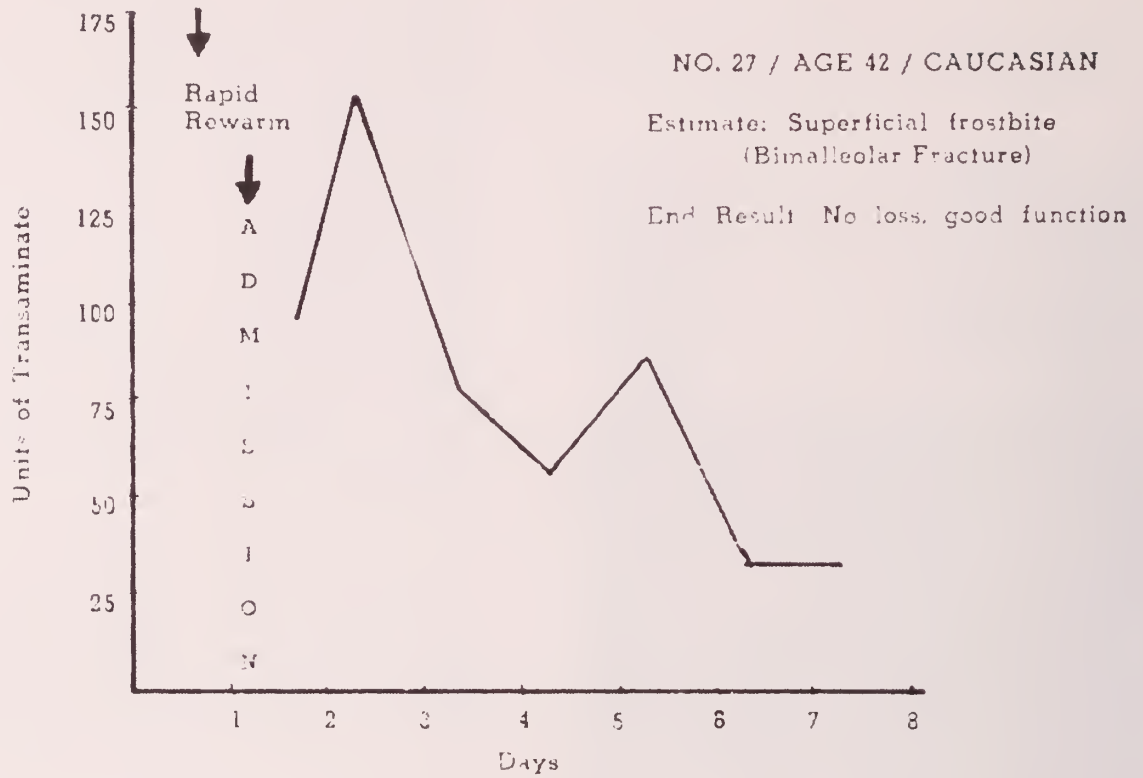
Some of the enzyme findings are quite interesting, and at this early stage appear to demonstrate, even with the non-specific enzymes, the following findings. There is a rapid rise and early fall in superficial injury or injury treated by rapid re-warming. These patients seldom have demonstrated any tissue loss.

Deep injury, treated especially by ice or snow pack or slow thawing, or re-warming at room temperatures, demonstrated a delay in enzyme rise, and a late fall that usually resulted in an anatomical pattern of marked tissue destruction. An interesting complication occurred in the plotting of enzyme levels when a combination of superficial injury of one extremity and deep injury of an-

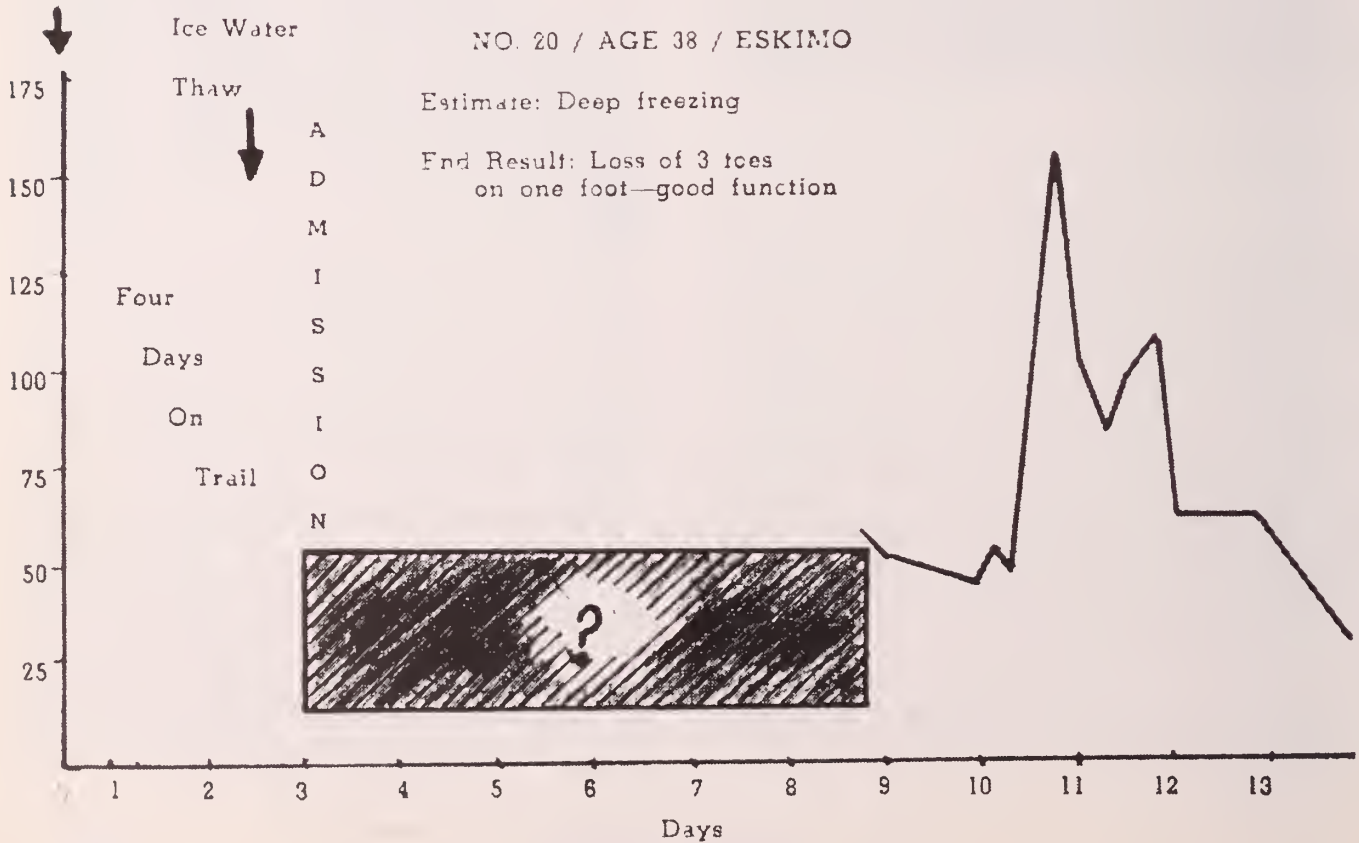


# POST FREEZING SERUM TRANSAMINASE

Insult -25 F Freeze



Freeze -50 F



other in the same individual was present. Then an early and late peak were demonstrated in the enzyme curve.

We have found, too, that in control subjects, with ultrasound an early rise occurs that evidently demonstrates tissue breakdown from utilization of ultrasound. When patients with deep injury were rewarmed by methods other than rapid rewarming, and had ultrasound as a part of their therapy, a dual peak occurred. Table I is presented, demonstrating a typical enzyme response in a patient who received rapid rewarming (#27) and one thawed utilizing ice and snow (#20).

### Radioisotope Studies

Patently, accurate, repetitive determinations of blood flow in cold-injured extremities could be invaluable in assessing degree of injury, prognosis and effects of therapy. Careful consideration of methodology suggested that radioisotope tracer techniques might well meet the problems of changing tissue resiliency, asepsis, and non-interference with arterial circulation, which practically exclude plethysmography, temperature measurement technique or angiography.

Throughout the past year pilot studies have been conducted to establish technique and to verify feasibility, utility, and safety. From studies on approximately twenty normal individuals it seems apparent that adequate counting rates can be obtained over the hand, fingers, forefoot, and toes.

Radioiodinated ( $I^{131}$ ) hippuran was administered intravenously. Dosage was 2 uc/kg body weight, total dose varying from 100-160 uc. The rate of appearance and the equilibrium level of radioactivity in the extremity was measured with a collimated scintillation detector coupled to a precision ratemeter and rectilinear recorder for readout. Only the equilibrium levels could be considered reproducible. Radiohippuran was rapidly excreted and in 24 hours only tracer amounts could be detected in the thyroid establishing the low biological half-life of the compound.

It is apparent from the work of others that several times the dosage described can be used with safety. The introduction of  $I^{125}$  into clinical

use will reduce expense and shielding problems. It is therefore concluded that semiquantitative measurement of circulation in the distal extremities and digits can be made safely with the use of radiohippuran as described and that this method may be applicable to the problem of cold injury.

### Summary

1. Fifty-one cases of frostbite are presented, treated by varying methods, and thawed by diverse means. In this small series the results of rapid rewarming in warm water, by warm packs or by whirlpool bath at temperatures of 110 to 118 degrees Fahrenheit, appear to demonstrate the most satisfactory results.

2. Regardless of method of thawing or rewarming, the results are more satisfactory utilizing whirlpool therapy throughout the course of treatment to those not receiving whirlpool bath and other physiotherapy.

3. Ultrasound as a form of deep tissue massage appears beneficial in cases of superficial injury, and at this moment in this small series, appears not beneficial and probably harmful in deep injury.

4. Of great interest to us are the results of enzyme studies, cursorily reported in this Third Part, that appear to provide us an early estimate of depth of injury and tissue changes, and some inkling of the ultimate prognosis even long before the gangrenous changes occur.

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# FROSTBITE AND HYPOTHERMIA - CURRENT CONCEPTS

## INTRODUCTION

W. J. Mills, Jr., M.D.

Prior to the Korean War, little information was available to the clinician treating frostbite and hypothermia, Alaska was a natural laboratory. The stimulation for investigating frostbite came as a result of a field clinic to a fishing community in Alaska where a group of patients were seen, all a crew of one vessel, who had sustained BK or AK amputation from frostbite and cold injury after shipwreck. This seemed such a large price to pay for this injury that effort was made to learn more of its pathogenesis and care.

Encouragement for further progress in the treatment of frostbite came about this time from supportive experimental reports using lab animals, from the papers of Lange<sup>3</sup>, Quintanella<sup>2</sup>, Fuhrman and Crismon<sup>6</sup>, and Meryman<sup>1</sup>, and others<sup>1</sup>. From others came suggestions that perhaps frostbite was a complex phenomenon including not only vascular change leading to gangrene, or mechanical damage to cells by ice, but dehydration of cells, protein denaturation, intra and extra cellular biochemical changes.

It soon became obvious that one of our early problems was prevention of infection, this latter complication causing as much tissue loss as loss of vessel patency. Soon after that demonstration, we were aware that even if anatomy was preserved by thawing methods or medical care, without the use of physiotherapy, small joint motion was lost from either immobility or cold arthritic changes in bone.

Impetus to the program of investigation was given in December 1956, by Dr. Peter Hamill, the District Medical Officer of the U.S.P.H.S. Hospital in Tanana. He had read of Meryman's work suggesting that rapid rewarming might be helpful, and Dr. Hamill then used this method to rewarm a patient with severe frostbite of feet and hands, hypothermia, all in a warm tub of 100° F.

Early in the investigation here, a contract\* from the Office of Naval Research was obtained that enabled increased clinical investigation at the Alaskan level. Throughout the literature there were

scattered reports of the use of rapid thawing but the results were said not to be good. Much of this poor result may have been due to failure to recognize the importance of avoidance of infection, restoration of motion, and the maintenance of circulation. Rapid rewarming has been an effective thawing method. This form of care received poor press and was in great disrepute as a result of the writings of Baron Larrey, Napoleon's chief physician during the Russian Campaign. In his memoirs, he wrote of the "congelation" effect, and the gangrenous changes that occurred after thawing. However, not for a hundred years was it appreciated that his method of rapid thawing was camp fire warming, therefore by excessive heat or temperatures greater than 150 to 170°, and that as demonstrated in this issue, the use of excessive heat is disastrous method of thawing and one where changes are irreversible and result in amputation at high levels. Further advances in the care of frostbite was advanced by Dr. Mundth who espoused the use of low molecular weight dextran. The summary of this method, as reported by Lt. Col. William Doolittle, will be presented in another issue of *Alaska Medicine*. Recent experimental work by Karow and Wells, regarding the alterations and extra cellular bound water, may prove helpful in our understanding of the pathogenesis of cold injury.

The Alaskan experience is a combined result of all the doctors and patients in our area, with special thanks to the aid of Dr. William Doolittle, Dr. Ed Lindig, and Dr. Petajan of the Fairbanks area, Dr. Robert Grossheim of the Alaska Native Hospital, Dr. Jack Roy of the Air Force Hospital at Elmendorf, with a note of special appreciation to Dr. Robert Whaley, Dr. Winthrop Fish, Dr. Rodman Wilson, Dr. Keith Brownsberger, Dr. Michael Hein, and Dr. Fred Hood, here in Anchorage, for all their interest and cooperation, and to Dr. Meryman and all of his colleagues in the laboratory who helped us put it all together.

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# FROSTBITE

## A DISCUSSION OF THE PROBLEM AND A REVIEW OF AN ALASKAN EXPERIENCE



**William J. Mills, Jr., M.D.**

*A Clinical Investigation under a contract  
NONR-3183(00)(NR105-249) between the Office  
of Naval Research, Department of the Navy, and  
William J. Mills, Jr., M.D.*

## INTRODUCTION

The importance of cold injury as a military medical problem is only too well known. Over one million cases have been recorded during the two World Wars and the Korean War, excluding Soviet and Chinese casualties. But, since cases may be scattered and escape compilation, it is not generally appreciated in this country, that cold injury is a significant medical problem during peace time as well, particularly in the subarctic areas of the world.

Two major categories of cold injury are recognized: non-freezing injuries of the immersion or trench-foot type, and the more acute freezing injury, properly designated as frostbite. Much of the cold injury seen during wartime is of the immersion foot, non-freezing type. Exposure is of many hours or days, and the result is an injury noted for its extensive edema, pain and slow recovery, and its long-lasting sequelae of pain, hyperhidrosis and sensitivity to cold. An additional late problem may be intermittent local ulceration, painful cutaneous fissuring, or chronic infection.

Most civilian injuries, on the other hand, result from a brief, more acute exposure to subfreezing temperatures. The sequence of events seen in a typical case of acute frostbite is as follows. Prior to thawing, the part is hard, cold, usually white and anesthetic, appearing solidly frozen, full thickness of the part, even in areas where freezing may actually be superficial. Thawing is often painful, particularly when accomplished rapidly. Delay in thawing is associated with less pain and may account for the popularity of ice, snow or ice water as a thawing media in many areas where self care is practiced. Following thawing, the part becomes flushed, often with an ominous purple hue.

The injured extremity is usually edematous with large serum-filled blisters developing an hour to several days following thawing. Unless accidentally broken, the blebs will remain intact until the fourth to tenth day when resorption of fluid begins and spontaneous rupture of the bleb may occur. As the blebs dry, a hard eschar develops on the injured surface. This eschar may be quite black, giving a false impression of deep gangrene. However, within three to four weeks, the eschar begins to separate spontaneously, revealing delicate but healthy epithelial tissue below.

When injury is so severe as to preclude tissue recovery, blebs are absent and the tissue remains cyanotic and cold. These areas, usually the distal phalanges, will show evidence of beginning mummification, often within a few days. Over a period of days, weeks or months, demarcation between healthy and dead tissue becomes more pronounced; the viable tissues separate and retract

from the mummified until spontaneous amputation of the soft tissue is essentially complete.

It must be emphasized that the foregoing description is based on circumstances uncomplicated by infection or surgical intervention. Infection or premature debridement may lead to unnecessary tissue loss, osteomyelitis with successively higher amputations, extensive skin grafting and prolonged hospitalization. The prevention of infection and the avoidance of unnecessary surgical intervention are essential for the preservation of the maximum amount of viable tissue.

The ultimate mechanism of tissue injury from freezing is unknown. However, it does appear that the extracellular ice crystals that form with the slow rates of freezing found in clinical frostbite do not cause mechanical injury sufficient to produce tissue death. The primary injury is presumed to be biochemical, resulting from the removal of water to form ice. Extreme dehydration is produced, with a concentration of solutes, particularly electrolytes.<sup>1</sup>

One of the most important effects of tissue freezing is the vascular stasis that develops following thawing. Quintanella, *et al*,<sup>2</sup> Lange, *et al*<sup>3</sup> and, more recently, Mundth,<sup>4</sup> have studied the microcirculation of experimental animals following freezing and thawing. Immediately following thawing the circulation is apparently unimpaired, but within a few minutes evidence of obstruction can be seen in the venules. Mundth reports that the obstruction appears to result from aggregations of platelets. Erythrocytes pile up behind them and stasis extends back through the capillary bed to the arterioles. Within two hours or less, there is complete irreversible stasis in the thawed tissue and the vessels are totally filled with a structureless, hyalinelike material. There is tissue edema and evidence of the extravasation of hemoglobin into the perivascular spaces.

On the basis of such observations, it was naturally assumed that much of the tissue loss in frostbite might well result from secondary vascular stasis and not from the primary effects of freezing. Earlier attempts to reverse or prevent the stasis were generally unsuccessful, although some clinical benefit was reported by Lange, *et al*<sup>5</sup> using continuous heparin infusions. No satisfactory evidence existed that tissue cells were not irreversibly damaged by the primary freezing injury and might fail to survive even if the circulation were maintained.

Mundth, *et al*<sup>4</sup> has investigated the use of low molecular weight Dextran following experimental freezing in rabbits. Observation of the microcirculation indicated that the Dextran did alleviate the post-thawing circulatory obstruction.



Infusion of this material as late as two hours following thawing markedly reduced tissue loss in frozen rabbit feet. This is presumptive evidence that some of the injury from freezing is not necessarily irreversible and that maintenance of the circulation may permit the recovery of tissue otherwise doomed.

A number of workers, particularly Fuhrman and Crismon in 1947,<sup>6</sup> demonstrated impressive results by thawing experimentally frozen animal extremities, in water at 42° C. These studies were repeated with varying success by others.<sup>2, 7, 8</sup> Although some results could be questioned on the basis of uncontrolled duration of exposure to the frozen state,<sup>1</sup> where this factor was controlled, there did appear to be a very real reduction in tissue loss following rapid thawing in experimental animals.<sup>8</sup>

A wide variety of clinical approaches to frostbite have been reported in the literature and one can find examples of almost every conceivable therapeutic measure. Attempts to assess various treatments usually suffer from lack of diagnostic criteria and an insufficient number of cases for statistical analysis. Much of the literature is concerned with problems of late treatment and sequelae. Of the promising early clinical procedures, one of the most encouraging has been that of Shumacker and associates<sup>9, 10</sup> who used sympathectomy to improve and maintain circulation.

A highly successful therapeutic routine has also been developed by Campbell<sup>11</sup> and is used by the International Alpine Rescue Commission. Thawing is achieved in a bath initially at about 50° F. (10° C.) and gradually increased in temperature, reaching 104° F. (40° C) in about thirty minutes. This technique is reported to prevent the pain of thawing. An open, dry procedure is recommended with limited debridement of bleb coverings after their rupture or collapse, and the application of silver or aluminum foil over granulating areas.

In Alaska prior to 1955, frostbite was treated by a variety of methods including rubbing with snow, thawing in an ice or snow bath, spontaneous thawing indoors, or rarely, immersion in warm fluid.<sup>18</sup> Subsequent care varied from total neglect to early surgery. Tissue loss was not uncommon and a distressing number of mid-foot and below-the-knee amputations had been reported. At this time, a series of studies was begun, in an effort to establish a uniform routine for both early and late treatment, to improve our own results and, in particular, to determine whether the beneficial results reported after rapid thawing of animal extremities could be duplicated in clinical injuries.

It is now possible to report on a series of two hundred (200) patients with frostbite, of whom one hundred and thirty (130) were treated

throughout according to a fixed therapeutic routine. Of this group, forty-six (46) were initially thawed in warm water. Sixty-eight (68) of the two hundred patients were treated by other methods that did not include rapid rewarming in warm water, or post thawing physiotherapy, whirlpool or open care. Two patients of this group of two hundred were immediately thawed with a warm water bath, but received other methods of post thaw care.

## METHOD OF TREATMENT

Patients seen prior to thawing of the extremity were at first thawed rapidly by immersion in well-agitated water maintained at between 100° and 112° F. (38° to 45° C.) Subsequent observations and second thoughts have resulted in a revision of the higher limit downward to 108° F. (42° C.) When thawing is conducted in the hospital, a whirlpool bath can be utilized for that purpose. Such rewarming is reserved only for patients seen prior to thawing. This treatment is considered useless and perhaps injurious to extremities no longer in the frozen state.

On admission, a frozen extremity appears white, yellow-white, or mottled blue-white, hard, cold, insensitive, and presents the illusion of being frozen solid. Even a relatively shallow freezing may give this appearance and an estimate of the depth and severity of the injury is impossible at this time. Early in our investigation we discarded the conventional terminology of first through fourth degree frostbite as a prognostic impossibility and have attempted only to describe the injury as superficial or deep. Even so, such an estimate may be changed by the method of thawing. A presumed deep injury, rapidly thawed, may assume the appearance of a superficial injury, while a superficial injury, thawed in ice or snow bath or by other delaying means, may then appear as a deep injury, the viability of underlying structures presumably having been jeopardized.

Immersion of the frozen extremity in the thawing bath is painful to the patient and sedatives or analgesics are used as indicated. As thawing proceeds, flushing will progress distally down the extremity. Thawing is continued until the flush has extended to the tips of the extremities and the immersion is then promptly terminated. The flushing may be an ominous purple in color despite the excellent results that can be anticipated following this mode of thawing. This burgundy hue is particularly associated with higher thawing temperatures. With rapid warming (100° to 112° F.) sensation returns to the affected part when thawing is complete. Sensation remains until the blebs develop and separate the surface layers. In no other method of thawing have we observed this.



Sensation remains absent for weeks when frozen tissues are allowed to thaw spontaneously in air or by delayed means such as in an ice or snow bath.

Following thawing, the patient is hospitalized with complete sterile precautions. The extremities are kept on sterile sheets under cradles. Sterile cotton pledgets are placed between the toes. The patient is kept in strict isolation with attendants masked and gowned. No dressings, ointments or other applications are used. Treatment is completely open. As blebs appear, every precaution is taken to avoid their rupture. In addition to the sterile precautions, infection is controlled by cleansing the extremity for twenty minutes twice daily in a whirlpool bath at 90° to 98° F. A mild disinfectant soap is used in the bath\*. Patients who have already thawed prior to admission are placed on the identical routine save that the initial thawing procedure is omitted.

Ideally, the blebs will remain intact and after five to ten days their serous contents begin to be resorbed. In some cases the blebs are accidentally ruptured and in other cases the patient may be admitted with blebs already ruptured. No attempt is made to debride. This is left to the motion of the separating tissues prematurely. If edges are trimmed, use care, avoiding damage to fragile tissues.

Of critical importance to the ultimate functional result is constant energetic active exercise of both large and small muscles and intermittent elevation of the extremity. As the blebs dry, hard, often black, eschars form. Where these prevent motion of joints they may be carefully split on the sides or dorsum of the digits. However, they are never removed but are allowed to be separated and gently debrided by the motion of the whirlpool. One of the many virtues of the whirlpool bath is the opportunity it affords for active exercise. The dried blebs and crusts are softened during the washing and the patients report greater ease and comfort of motion. The importance of active joint motion cannot be over-emphasized, particularly for the prevention of flexion-contractions of the digits, not an uncommon sequela of frostbite even when complete anatomical preservation is achieved.

The use of water and whirlpool is often criticized on the basis that it is a "wet" regimen, a total misunderstanding of its use since, within minutes of removal from the whirlpool bath, the parts are dry and exposed to the air under a protective cradle.

We have been greatly impressed by the ability of the whirlpool bath to cleanse gently, to debride, to promote circulation, to permit and encourage active motion and to impart a sense of well-being to the patient. We have been particularly impressed

with the efficiency with which whirlpool controls infection, even in cases that have come to us complicated by maceration and advanced wet gangrene. We wish to emphasize that whirlpool therapy is a central element in our therapeutic regime and responsible, in our opinion, for much of our success in minimizing tissue loss following severe injuries.

Antibiotics are rarely necessary save for deep infection, again because of whirlpool lavage of superficial structures. No narcotics are used in uncomplicated cases after initial thawing. Anti-tetanus therapy is used where indicated by associated trauma. Special problems related to concomitant fracture or other special situations will be discussed subsequently.

When the eschar has begun to slough in the whirlpool and healing is clearly on the way, sterile precautions are discontinued but whirlpool and active exercises are rigidly pursued. Where distal parts of the extremity are to be lost and the part remains black and cold, often no debridement is done until spontaneous amputation of the soft tissue is virtually complete. This may require anywhere from three weeks to four months. The mummified portion may then be surgically removed without danger of retraction, infection or the necessity of skin grafting or subsequent revision of the stump.

The therapeutic regime, in summary, consists of *rapid thawing where possible, avoidance of infection, whirlpool, continual active exercise* with periodic elevation, *prohibition of surgical debridement* and *postponement of surgical intervention* pending complete demarcation, with spontaneous amputation of soft tissues.

## RESULTS

The first 200 patients with a total of 413 cold-injured extremities have been treated according to the foregoing procedures. Evaluation of 500 patients is in process now, generally confirming these initial impressions. Cases of wet-cold, non-freezing injury have been excluded from this series. The first 51 patients treated have been reported in detail in a previous paper<sup>12</sup> and Washburn<sup>13</sup> has summarized our methods and several of our cases in an article on arctic problems published in the *New England Journal of Medicine*.<sup>14</sup>

Probably the single greatest obstacle to a comparative analysis of frostbite therapy is the impossibility of making any quantitative assessment of the extent of injury prior to the institution of early therapy. In the past, all criteria by which frostbite was partitioned into degrees of injury have been based on ultimate outcome and tissue loss, even though theoretically used in initial

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\* We have used pHisoHex and Betadine.

diagnosis. In fact, no method or set of criteria exist for evaluating the injury prior to thawing. Histories are totally unreliable since freezing is generally insidious and clothing and special contributing factors prevent direct correlation of temperature and wind conditions with the severity of the injury. Thus the direct comparison of individual clinical cases is meaningless, placing greater burden on clinical impression and requiring the assembly for statistical analysis of a larger number of cases than would be necessary for the study of a more quantifiable disease.

No attempt, therefore, has been made to estimate degree of injury prior to therapy. Cases have simply been evaluated on the basis of end result, both functional and anatomical. Table I explains the classifications used. Where a single individual has had multiple extremities involved, he has been classified on the basis of the most severely injured. Table II compares the results obtained in patients whose frostbitten extremities were treated throughout by our standard program including rapid thawing, with those receiving the same standard program without rapid thawing; those treated by others, by other methods, and only seen by us following the resolution of their frostbite; and with two cases which were rapidly thawed but treated by others by methods other than our standard routine. Table III illustrates the results achieved when injuries treated by our standard routine are compared on the basis of the method of thawing. Both functionally and anatomically the percentages of extremities with good to excellent outcome are nearly identical regardless of whether extremities received rapid or slow thawing. Although slowly thawed extremities apparently suffered more tissue loss than those rapidly thawed, this is in part explained by the fact that some of these suffered refreezing or were thawed by methods felt to be deleterious such as by ice and snow and excessive heat. Those extremities receiving neither rapid thawing nor our standard routine showed a very high percentage of tissue loss and major amputation, in our opinion largely due to uncontrolled infection and premature surgery.

Table IV illustrates the distribution of injury produced by refreezing or by deleterious methods of thawing. The preponderance of poor results is evident.

Table V illustrates the influence of infection or subsequent results. Only two of the cases without infection lost tissue, while 25 of those with superficial infection and 29 of those with deep infection did. Seven patients in the "E" category, and one in the "F" category, received whirlpool therapy.

## EVALUATION OF SYMPATHECTOMY

We have studied the results of post-thaw lumbar and cervical sympathectomy in patients with apparent bilaterally similar injury to hands or feet, or both. The sympathectomy has been performed on one side only, utilizing the untreated extremity as a control. Eventual contralateral sympathectomy may follow.

Unilateral ganglionectomy has been performed as early as twelve hours and as late as three weeks in the acute cases. The results to date are interesting, somewhat different than expected, and quite definite. The effects of sympathectomy have been assayed on the basis of recordings of tissue temperature using thermistors superficially, intramuscularly and subcutaneously placed. Our results to date are somewhat similar to those reported by Isaacson and Harrell.<sup>15</sup> In almost all cases our patient was happy to have had the sympathectomy performed. This was so until approximately six months to one year had elapsed. Complaints then were often that the ganglionectomized extremity was "too dry". Regardless of the time of the procedure, relative to the date of thawing, it did not appear to preserve tissue. In fact, the extremity not subjected to ganglionectomy often showed more preservation of tissue.

However, there were favorable results which appeared to be associated with the procedure. Infection, if present, superficial, or deep, was almost always rapidly resolved, often within a forty-eight hour period. Edema rapidly diminished, and pain was usually much less and often completely disappeared on the operated side. Hyperhidrosis soon disappeared and sensation appeared to return earlier on the sympathectomized side. We noted, as previously reported, that demarcation of the tissue appeared more rapidly on the side of the sympathectomy, but at this time cannot determine whether that in itself is beneficial. This more rapid demarcation may not indicate more ready healing, but instead, more intense and immediate vascular shunting with more rapid necrosis and mummification of the part distal to the shunt.

## SILVER NITRATE

Over the past six years, after the method of Moyer, et al,<sup>16</sup> for burns. 0.5% silver nitrate has been intermittently lavaged over the area of frostbite using a contralateral hand or foot as control whenever the depth of injury appeared to be bilaterally similar. The end result has been comparable to that following the use of other soaks or cleansing agents; the epithelialization is similar whether skin or scar, but there is perhaps



one outstanding effect noted. Pain is less, and infection, even superficial, appears less obvious. The handling of Ag NO<sub>3</sub>, staining of bed linen, floors and hospital equipment by the silver nitrate is a mild aggravation making this method of care unpopular with Hospital Administrators and Nursing Supervisors. This problem is somewhat eased by the use of disposable supplies.

### SKIN GRAFT PROCEDURES

To hasten healing, skin grafts have been utilized between the third to the twenty-first day and as a reconstructive procedure at any time thereafter. Split thickness skin and Reverdin pinch grafts have been well received during the early stages of healing if the part to which the graft was applied was rapidly thawed. The split thickness and minute grafts have been irregularly successful when applied to extremities spontaneously thawed or thawed by delayed means with ice or snow. After three months or longer, all types of graft appeared effective in the absence of infection, once the denuded areas were well vascularized.

Split thickness skin cover in frostbite often requires intermittent saline soaks to prevent maceration and infection. The procedure of grafting is worth a trial in many cases, often preventing exposure and necrosis of tendons, underlying fascia, and joint and periarticular structures. The skin graft procedure is benign, and can be performed without difficulty under local anesthesia. Care post grafting must be directed to maintaining a clean graft surface, sometimes difficult because of the previous necrotic bed upon which the new graft rests.

### HYPOTHERMIA AND DEHYDRATION

Death from general body cooling is not uncommon in the arctic and subarctic. In the past three years we have had the opportunity to see and treat four patients in deep hypothermia. Only one, the last treated, survived. Since that time, twelve patients with varying degrees of deep hypothermia have been treated without loss of life, although preservation of anatomy when hypothermia is accompanied by frostbite, is more difficult than the essentially normothermic patient with freezing injury.

All patients had sustained freezing of the extremities as well as general body cooling. Sudden accidental cooling of the body and depression of the normal core temperature is serious and often fatal. At rectal temperatures below 92° F. homothermic control becomes unstable, and, if cooling persists, may be lost altogether. Coma often develops early and cardiorespiratory failure may occur even above 88°F.

We recommend, in this mixed problem, the same resuscitation techniques which are followed when general hypothermia is used as an adjunct to surgical anesthesia. These techniques include rapid warming with warm packs and blankets, intravenous infusion of glucose and water, and respiratory aids including intubation and oxygen if necessary. The possible need for cardiac defibrillation or tracheostomy should be anticipated. The process of recovery is often complicated by pre-existing anoxia, trauma with excessive blood loss, and alcoholic stupor.

Rapid warming in a water bath at 90° to 100° F. brings the patient quietly to a responsive state, alert and rational. However, the release of accumulated acid end products of metabolism can create a sudden metabolic acidosis with death by ventricular fibrillation in less than two to three hours following rewarming. Following slow spontaneous, or delayed rewarming, death may occur as much as twenty-four to forty-eight hours later.

It is essential that one or more intravenous routes be established in the hypothermic patients to enable electrolyte control with the determination of, at least, pH, pCO<sub>2</sub> and pO<sub>2</sub> and the administration as required of buffering agents such as sodium bicarbonate or THAM. EKG monitoring is indicated in view of the hazard of ventricular fibrillation. In rare instances the patient may be in alkalosis rather than acidosis as a result of gastric suction or vomiting or from the retention of potassium or sodium.

The irrational behaviour previously reported<sup>1,2</sup> in victims of freezing injury may actually represent post-traumatic phenomena secondary to pre-existing effects of water deprivation and dehydration. This behaviour is particularly likely in the survival victim rescued after days of wilderness exposure, often without food and water.

### X-RAY EVALUATION OF EXTREMITIES

The roentgenographic examination of the involved extremity is usually negative when performed in the first ninety days, unless severe infection with osteomyelitis is present or amputation has occurred. Occasionally in the absence of a strenuous program of physiotherapy and digital exercises there may be osteoporosis of the involved digits and tarsi or carpal bones.

Between the third and sixth months, apparently related more to the depth of injury than the method of thawing, there appears fine, irregular lytic areas, generally in the metacarpal or metatarsal phalangeal or proximal or distal interphalangeal joint areas (Fig. A). These punctate lesions are often juxta- or subarticular and



occasionally extend into the joint space, usually a late occurrence. In most of the cases examined these punctate lesions may worsen over one to three years and then many may eventually decrease in size or fill completely. Our experience is similar to that reported by Vincent, *et al*,<sup>14</sup> who found no pattern for this change.

Biopsy of these areas on the few occasions permitted, demonstrated the lesions to consist of dense fibrous connective tissue, suggesting areas of chronic inflammation. Prominent vascular channels are seen on some sections in the indentations that extend into the subarticular tissues. These changes are somewhat similar to the "punched out" lesions of rheumatoid arthritis or gout. The soft tissues are fusiform and obviously swollen. The area most commonly involved in our series is that of the interphalangeal junction, usually the proximal interphalangeal joint, with the lesion most often at or near the articular margins. These X-ray changes are often associated with the apparent loss of the volar fat pads subcutaneous fat the glossy appearance of skin over the digits, fusiform enlargement and contracture of the involved joints.

The changes seen in this group of patients appeared earliest at six weeks in a patient thawed

by rapid rewarming, and latest at eighteen months in a patient likewise rapidly thawed. Possible causes of the lesions are (1) disuse, (2) direct effect of cold, or (3) avascular necrosis as a result of thrombosis of the articular branches of the volar or dorsal digital arteries. Although there is no direct evidence, it is thought that the lesion is an effect of thrombosis or blockage of the articular branch of the digital vessels, with further loss of nutriment to the subarticular cartilage.

TABLE I

Classification of degree of ultimate injury

- A No recorded or demonstrated residual
- B 1) Dysesthesia  
2) Intrinsic muscle atrophy
- 3) 3) Skin loss requiring skin grafting
- 4) 4) Volar fat pad (digital) atrophy or loss
- 5) 5) Limitation of joint motion
- 6) 6) Neurovascular sequelae (hyperhidrosis, hypesthesia, hyperesthesia, paresthesia, pain)
- C Amputation, distal phalan any level, any number
- D Mid or proximal phalangeal amputation, any level any number
- E Complete phalangeal loss at metacarpo-phalangeal or metatarso-phalangeal
- F Major amputation of an extremity

TABLE II

Results of varying methods of treatment.  
(Figures are percentage of patients in each category and figures in parentheses are numbers of patients)

	Category of Injury												
	A		B		C		D		E		F		Total Patients
Full frostbite program (rapid thawing, whirlpool, P.T., open care)	9	(4)	87	(40)	0		4*	(2)	0		0		(46)
Program as above except thawing by other than rapid	8	(7)	70	(58)	6	(5)	7	(6)	8	(7)	1	(1)	(84)
Other treatment programs not including P.T., whirlpool, or open care	9	(6)	39	(27)	12	(8)	12	(8)	9	(6)	19	(13)	(68)
Rapid thawing but with other treatment programs	50	(1)	50	(1)	0		0		0		0		( 2)
All patients	9	(18)	63	(126)	6.5	(13)	8	(16)	6.5	(13)	7	(14)	(200)

\*Case No. 19: 1st case of Rapid Rewarming by H<sub>2</sub>O (100°F.) Loss due to premature surgical debridement. Also case No. 77: Loss of 5th finger complete following laceration of left wrist (ulnar arter incised) and laceration left antecubital fossa in suicide attempt prior to freezing episode (as part of suicide attempt).

TABLE III

Results of Standard Frostbite Program Applied to Patients with Injuries Thawed by Rapid,  
Slow or Delayed Means or by Excessive Heat  
(Data presented as percentage of patients in each category. Figures in  
parentheses are number of patients)

	Category of Injury												Total Patients
	A		B		C		D		E		F		
Rapid thawing in warm bath 100°-115° F.	10	(5)	86	(41)	0		4	(2)	0		0		(48)
Slow thawing, predominantly spontaneous in air at room temperature	9	(12)	61	(81)	8	(11)	6	(8)	5	(7)	10	(13)	(132)
Delayed thawing by ice, snow, or cold water bath	6	(1)	25	(4)	13	(2)	25	(4)	31	(5)	0		(16)
Thawed by excessive heat	0		0		0		50	(2)	25	(1)	25	(1)	( 4)
All patients	9	(18)	63	(126)	6.5	(13)	8	(16)	6.5	(13)	7	(14)	(200)

TABLE IV

Results of Mixed Insult Including  
Refreezing and Thawing with Excessive Heat.  
(Figures indicate numbers of patients)

	Category of Injury						
	A	B	C	D	E	F	Total
Refreeze (freeze-thaw-freeze, thaw by any means)	0	0	0	2	2	0	4
Immersion injury followed by freeze, thaw by any means	0	1*	0	0	1	7***	9
Fracture plus freeze	0	5***	0	0	0	3	8
Thaw by excessive heat (above 120° F.)	0	0	0	2	1	1	4
TOTAL	0	6	0	4	4	11	25

\* Age 12, spontaneous thaw.

\*\* Includes one patient, diagnosis diastasis tibia, fibula (distal), thawed spontaneously; one patient diagnosis metacarpal fracture, thawed spontaneously; two patients fracture of tibia, rapidly thawed; one fracture ulna, undisplaced, thawed spontaneously.

\*\*\* Exposure in all cases 5-7 days minimum.

TABLE V

Result Related to the Presence or Absence of Infection

		Result Related to the Presence or Absence of Infection												Total
		A		B		C		D		E		F		
None		16	(11)	81	(57)	3	(2)	0		0		0		( 70)
Superficial Infection		7	(7)	68	(68)	7	(7)	10	(10)	6	(6)	2	(2)	(100)
Deep Infection		0		3	(1)	13	(4)	20	(6)	23	(7)	40	(12)	( 30)
TOTAL		9	(18)	63	(126)	65	(13)	8	(16)	65	(13)	7	(14)	(200)



## DISCUSSION

Evaluation of the effectiveness of rapid thawing or any other therapeutic procedure is hampered by the absence of diagnostic criteria of the severity of the initial injury. Initially in this study it was attempted to estimate the degree of injury on admission of the patient, but even when the estimate was limited to only two classifications, superficial or deep, twenty-one of the first fifty-one cases were found to have been erroneously classified when viewed in retrospect. Furthermore, it was found that early classification had no clinical usefulness since there was no evidence that any variety in treatment would be indicated on the basis of an estimate of the severity of injury.

Because of the absence of initial quantitative diagnosis, this data is presented solely on the basis of ultimate outcome. This is most unsatisfactory because of unique circumstances in individual cases. In Table II, for example, cases in D and E categories receiving routine treatment, but not rapidly rewarmed, were almost all thawed by either excessive heat, ice and snow, or suffered a freeze, thaw, refreeze injury, events known to aggravate the cold injury. Half of the cases receiving major amputation after "other" treatment suffered shipwreck and exposure to cold water immersion for several days with superimposed freezing. Although our cases are certainly random, wide variation in circumstances and depth of injury makes even the attempt to evaluate treatment on the basis of final outcome most uncertain despite the relatively large number of cases reported.

Regardless of the difficulties in interpreting the statistical data, there is a definite clinical impression of the superiority of rapid thawing. Extremities rapidly thawed show flushing to the distal tufts of digits and the rapid appearance of normal pink color. Sensation to pin-prick is always present after rapid thawing until separation of blebs begins. Sensation was never found following other modes of thawing save in the most superficial injuries which do not go on to bleb formation. After rapid thawing blebs appear in one to eight hours. They are larger and more distal than after other thawing methods and are filled with clear serous fluid, never bloody. Bleb appearance may be delayed for two or three days after spontaneous thawing, seven or eight days after ice and snow thawing. These latter thawing methods produce smaller blebs which are often sero-sanguinous or black in color.

The hospital course appears smoother and shorter for the rapidly thawed extremities. Patients generally can be discharged in six weeks or less. Rapidly thawed extremities dry sooner, have less superficial infection, and few complications.

Recently, emboldened by past success, we have discharged our patients after bleb rupture and eschar formation, to be followed at home. This early discharge to home care may be from third to twenty-first day, depending upon the severity of the cold insult. There the "clean" hospital regimen is employed during the Jacuzzi home whirlpool, or an oscillating washing machine, even available gas operated in remote areas. There was an attempt, in analyzing this series, to correlate hospital days with thawing and treatment methods but we found this impractical because of the intrusion of other factors. An early amputation, for example, results in a shorter hospitalization than a protracted effort to save the maximum tissue. Indigents with no home care convalesce in the hospital and patients in the Public Health Service Hospitals serving the Alaska native population may stay longer than those in private facilities. Discharge was often delayed pending the availability of transportation to distant or inaccessible homes.

It is felt that there is little question regarding the total destruction produced by freezing, thawing, and refreezing. These tissues become black, dry and shriveled or liquefy completely within seven to nine days with no evidence of viability at any time. Slightly less rapid development of gangrene is also seen in severe injury following ice and snow thawing or with concomittant fracture which interferes with circulation in the extremity. The superposition of a burn over cold injury from the use of uncontrolled dry or wet heat for thawing is self-evidently destructive.

In our Alaskan experience, poor prognostic signs during early treatment include the late appearance of small, dark colored blebs; the failure of blebs to extend down to the tips of the volar pads of the digits; cold cyanotic distal parts and, of course, obvious mummification. Good prognostic signs after thawing; include sensation to pin-prick, good color, warm tissues, large clear blebs appearing early and extending to the tips of the digits.

Based on comparisons of the results obtained with random cases, it is felt that the clean conservative routine herein described has produced substantially less tissue loss and functional disability than the various procedures previously used. In particular, the absence of amputations higher than the metacarpal or matatarsal-phalangeal joint in any patients receiving this routine treatment compares favorably with the periodic mid-foot, below and above knee amputations previously seen throughout Alaska. The clinical impression is that substantial benefit is incurred by rapid thawing. This method will be continued as a recommendation for treatment, wherever

extremities are seen prior to thawing. It is anticipated that the collection of increasing numbers of cases treated by rapid thawing will provide a stronger statistical base from which to draw more positive conclusions in the future.

### SUMMARY

1. Two hundred cases of frostbite have been treated according to a standard hospital routine consisting of open, sterile care, twice daily whirlpool bath, intensive active physiotherapy, and postponement of surgical intervention. Twenty-one patients, or 10.5% lost phalanges or portions of phalanges. There was one major amputation. Seventy additional cases with frostbitten extremities received other forms of treatment. Of these, thirty-five, or 50%, lost tissue. Thirteen patients, or 18.57%, had major amputations.
2. Of the cases treated, forty-eight were seen prior to thawing and were rewarmed in water at above body temperature. Two extremities lost a digit and one the tuft of the distal phalanx. Although the number of cases is insufficient to permit positive conclusions, the clinical results following rapid thawing appear to be superior to those following other means of thawing.
3. The follow period of this report ranges from eighteen (18) to five (5) years.



FIG. A

*Xray of digits, severe freezing, three years previously. Thawing was by rapid rewarming in warm water. On this section of film is the fusiform enlargement of the pip joints and the sub and intra articular lytic areas.*

### CASE HISTORIES

The following case histories are illustrative of the sequence of events that may be anticipated in typical severe frostbite treated by this standard routine.

#### CASE NO. 187, EXCESSIVE HEAT

This seventy year old fisherman, hardy, and inured to Alaskan weather and winters, was exposed for four or five hours, winds fifteen knots, temperature -17°F., lost on a trail. He eventually was forced to crawl on hands and knees and was discovered by a nearby cabin dweller alerted by a barking dog. The patient was brought into a warm area, hands and arms were described as being in the full fist position, hard, frozen, white, and similar to two clubs. They were without sensation or motion. Thawing was begun with snow and ice water, and when this failed to rapidly thaw the part, the well-meaning first aid was applied by pouring scalding water over the frozen extremities until thawing occurred. The result was burn over previously frozen parts. (Fig. 1 - 4).

#### CASE NO. 91: REFREEZE (Fig. 5 - 8).

This adult mountain climber, age thirty-one, sustained injury over a period of several days at temperatures from -18° F. to -35° F., wind fifteen to twenty knots, altitude approximately 17,000 to 18,200 feet. While attempting direct ascent to the summit at about the 18,000 foot level, boots in place twelve hours, he noted loss of sensation and eventually upon returning to his tent found his toes white, immobile, with the resolution of what appeared to be at least superficial freezing injury following massage and warming of extremities by the accepted mountaineers method of using heat from his companion. Another attempt was made several days later, this time successfully, the summit at 20,300 feet was attained, during which he had complete loss of sensation. Upon reaching the 17,000 foot level and again thawing the extremities, it was noted that there was no evidence of pain, but a gravelly sensation in the heel and the development of small dark serosanguinous blebs distal to the metatarsal phalangeal junctions. Temperatures on the second attempt had been in the range of -30° F., wind at forty knots. On the second descent, after the second freezing insult, obvious demarcation had been identified at the mid-arch.

Eventually the climber descended to the 14,000 foot level, and there was met by rescue aircraft. He was admitted to an Anchorage Hospital, and placed on the standard frostbite



program during which he suffered rapid destruction of tissue commonly seen following this type of injury.

#### **CASE NO. 4: SPONTANEOUS THAWING**

This patient, a seventy-five year old fisherman and trapper, was injured when out at extremely low temperatures, moderate wind, deep snow. Exposure time may have been as long as twelve to fourteen hours, the entire episode complicated by alcoholic excess. He crawled through deep snow drifts, eventually reached his own cabin where his badly frozen extremities thawed spontaneously. Following admission to a regional hospital, he was placed on a standard regime as described in this article. His case history is representative of the severe changes following spontaneous thawing, other patients with spontaneous thawing having had minimal or no loss. As with delayed thawing, the results of spontaneous thawing are unpredictable. When examined his radial pulse was adequate. His hospital course after three weeks resulted in demarcation at the mid-proximal phalangeal area, complete demarcation developing at approximately six weeks.

#### **CASE NO. 78: RAPID THAWING**

This fourteen year old boy went hunting, walking twelve miles during his trip. The ambient temperature was -20°F., with little wind. He was wearing borrowed tight leather boots, covered with overshoes. One hour after leaving home his feet were noticeably cold and painful. Three hours later sensation was absent completely, the feet painless, and he continued hunting without pain, and therefore more comfortable. Six hours later he arrived home, overshoes filled with snow, and frozen to the boots. Foot gear was removed with difficulty, exposing cold, rigid, pale, yellow-white discolored feet, "solid to the ankle". He was taken to the nearest hospital. Examination there demonstrating an anesthetic foot, digits still in the "frozen" state, with collapse and compression of the volar pads. Forty-five minutes had elapsed since removal of the shoes and by this time a purple-red line of discoloration had developed at the line of demarcation between rigid and softer proximal tissues. The area involved was without sensation or motion.

The feet were thawed in water, temperature 110°F. until flushing of the distal pads was evident. The resulting "burgundy wine" hue of the distal foot persisted until the gradual development of blebs. His course was satisfactory, (Fig. 17) and he was discharged from the hospital for home care three weeks after injury. This represents a rapid hospital course for what was probably a severe

injury, in our opinion, possibly only with rapid rewarming techniques.

#### **CASE NO. 135:**

#### **FREEZING INJURY AND EARLY SYMPATHECTOMY**

Brought down after Helicopter engine failure in mid-winter on the Arctic Coast, this pilot walked out for help. He traveled for five hours, wearing felt 'bunny' boots, and lost sensation in his feet at the end of that time. Wind approximately 10 knots, temperature -30° F. He continued walking for another 7 hours and was finally rescued by another aircraft. He had minimal thawing by engine heat and was still demonstrating frozen tissues in the distal foot upon arrival at the Pt. Barrow Hospital. There he was thawed in a warm tub, 108° F., and within a few hours developed return of sensation and large distal blebs. His treatment was the regimen described in this paper, with the added procedure of lumbar sympathectomy on the left.

#### **CASE NO. 7:**

#### **DELAYED THAWING BY ICE AND SNOW**

This case represents a very popular method of thawing the frozen extremity all over the world in the frigid zones — that of immersion of the limb in ice water or a snow bath. This is popular presumable because the thawing is quite painless. The patient, a 54-year-old Eskimo, a trapper and hunter, was on the trail when a blizzard struck. His dog-team ran off, leaving him without food, water, or shelter, with the ambient temperatures between -50° F. and -20° F. He walked for six days, with no problem in his animal skin 'mukluk' footgear until the latter part of the second day, when he lost sensation in his toes. His feet were "frozen solid" by the third day. He walked into the Arctic village of Black River on the evening of the sixth day, having hiked an average of twenty to thirty miles daily, with only snow as oral fluid intake. His feet were frozen for three to four days, according to history and he left them so without attempting to thaw the extremities in order to maintain them in the solid state for walking and to survive. Upon reaching the village, his feet were immersed in snow and ice water, and thawed by delay over a period of eight hours.

#### **CASE NO. 45:**

#### **EXTREMITY FRACTURE FOLLOWED BY FREEZING AND SPONTANEOUS THAWING**

This patient was on a caribou hunt in the Northwest coast area of Alaska when his small plane collided with a mountain at the 5,000 foot



level. He sustained a fracture of the tibia, a fracture of the talus and was unconscious. His companion, equally severely injured, including a fracture of his lumbar spine, crawled 13 hours down to a settlement and eventually arrived at an Alaska Native Hospital. His thawing was in transport and was spontaneous. Temperatures in the area of the accident were well below zero and the winds were between 25 and 30 knots.

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## APPENDIX

# WIND CHILL - AND COOLING

In 1945, Dr. Paul A. Siple<sup>1</sup>, famed for his physiological observations in the Antarctic and a member of the Antarctic Byrd Expeditions, published his paper on *Measurements of Dry Atmospheric Cooling in Subfreezing Temperatures* in the Proceedings of the American Philosophical Society. From his original formula that took into account the combined cooling effects of temperature and wind speed, there has developed a wind chill chart so familiar to many of us. The chart published here has been produced by the Alaskan Command, U.S. Air Force, and is reproduced with their permission. This chart, and others like it, establish within fairly accurate bounds, the ambient unmoving air temperature that would be required in order to produce the same effect on exposed flesh, that a particular wind and temperature combination might produce.

Almost all Alaskans know that the increased velocity of wind may cause increased danger of skin freezing. Many assume that this increase in wind velocity causes the ambient air temperature to fall lower as the wind velocity rises. This is untrue. What does occur, is the phenomenon of air

movement, so that warm or warmed air is moved away from the individual exposed to the wind, causing first local then general body cooling. Any resultant decrease of skin temperature is due to heat loss, insidious or sudden, causing local vasoconstriction, vascular shunting and cellular changes until eventually ice forms in the tissues, with true tissue freezing or frostbite. This phenomenon can be readily proved. Merely place a laboratory recording thermometer with a thermistor attached (or any outdoor thermometer), out your car window, at temperatures in the neighborhood of 0° to -20° F., just a nice winter day in Anchorage, Alaska. Let it sit for a few minutes until the temperature reading has stabilized. This temperature, as read, will remain at the ambient air temperature level, such as -20° F. Now slowly accelerate your vehicle to 60 miles per hour; the temperature remains unchanged still at -20° F. Now attach the thermistor to a bared hand. Place your ungloved hand out the same car window in the same ambient temperatures, in this case we have chosen -20° F. at 0 miles per hour, the skin temperature may be

read from the resting state at approximately 93° F. or in that neighborhood, (normal skin temperature in the non-smoker) and it will slowly drop as heat is lost to the exterior, falling sometimes as low as 85° to 80° F. very rapidly. As the car is accelerated and the protective air layer is moved away, the thermistor records continued skin heat loss or skin temperature fall; if this is continued skin temperature may drop to a level near 23° F., the temperature level where freezing of skin may actually occur.

Cooling then, by one definition, may be described as loss of heat. Freezing is such total heat loss that ice forms in the exposed tissues.

This effect of the cooling power of the atmosphere (by wind) is primarily heat transfer by convection — in human cases, by exposure of uncovered flesh to the environment. Wind therefore has an important effect on body temperature by it's influence on air cover and air movement over the exposed skin. Even small amounts of air movement have considerable chilling effect because this movement disrupts or removes the thin layer of warmed air that builds up near and about the body. This air movement leads to loss of total heat, since heat is transferred from the core of the body to rewarm the new colder air, replacing that blown away. Therefore, wind chill leads not only to frostbite locally, but may contribute to general hypothermia. Wind also has not only a chilling effect, but robs any insulation of it's effectiveness if the insulation becomes wet, by combining heat loss of convection with that of loss due to conduction.

Heat is transferred (or lost) only when there is a temperature gradient; the smaller the difference between the bodies measured, the less the loss. Avoidance of heat loss to wind (air movement) is best by insulation, the secret being that the insulating layer keeps the outer face at the environmental temperature, and the inner face of the insulating area, that at the skin surface, at normal skin temperature, so that there is little loss or transfer of heat, between the two surfaces. The insulator serves to keep heat from escaping from the body to the cold environment. This insulator is the same in either case, namely air.

Warm air that is in small compartments, not moved rapidly about, weighs very little. This is the advantage of fishnet underclothing; a layer of air is trapped and cannot be dissipated by wind or air movement. In insulation then, it is the thickness of the insulator, not the weight, that is important. One is able to make up for the loss of heavy, tightly woven material, by the addition of a light, thin, outer wind proof shell. This further maintains the effectiveness of the layered principle and the utilization of light trapped air.

Wind chill may occur not only from natural wind, but also with air movement generated by automobile, snowmobile, aircraft or helicopter rotoblade. These vehicles generating their own air movement (or wind) may predispose to frostbite or general hypothermia in those poorly covered.

The basic problem then is that of heat loss or its prevention. Each individual has his own private inner climate<sup>2</sup> that floats about in the general outer climate of the natural elements. This inner climate is protected by an insulating shelter, out of wind, or clothing, and the thin layer of air. The metabolic furnace of the individual is fueled by calories; some of these calories are stored, and some are used as fuel to keep the inner climate temperature at near 98.6° F. Realizing this, and realizing that skin freezes at approximately 20° to 24° F. or -5° to -6° C., if you can avoid those temperature levels, your tissues cannot freeze. Realizing that heat is transferred from a warm to a cold environment, and that if your body is losing heat, it must be to a colder exterior region, then if you have a sensation of being cold or cooling, when previously you were warm, you must have a leak in the protective physiological system. Your inner climate needs adjusting or your furnace is in need of repair. By redeveloping your protective air cover, replenishing your caloric reserve, with the addition of water, you may stabilize your own inner climate. And if you know all of this, or more, you will no longer fear the outdoors in winter, wind or not.



*Gone with the wind*

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WIND SPEED		COOLING POWER OF WIND EXPRESSED AS "EQUIVALENT CHILL TEMPERATURE"																					
KNOTS	MPH	TEMPERATURE (°F)																					
CALM	CALM	40	35	30	25	20	15	10	5	0	-5	-10	-15	-20	-25	-30	-35	-40	-45	-50	-55	-60	
		EQUIVALENT CHILL TEMPERATURE																					
3 - 6	5	35	30	25	20	15	10	5	0	-5	-10	-15	-20	-25	-30	-35	-40	-45	-50	-55	-60	-70	
7 - 10	10	30	20	15	10	5	0	-10	-15	-20	-25	-35	-40	-45	-50	-60	-65	-70	-75	-80	-90	-100	
11 - 15	15	25	15	10	0	-5	-10	-20	-25	-30	-40	-45	-50	-60	-65	-70	-80	-85	-90	-100	-105	-115	
16 - 19	20	20	10	5	0	-10	-15	-25	-30	-35	-45	-50	-60	-65	-75	-80	-85	-95	-100	-110	-115	-120	
20 - 23	25	15	10	0	-5	-15	-20	-30	-35	-45	-50	-60	-65	-75	-80	-90	-95	-105	-110	-120	-125	-130	
24 - 28	30	10	5	0	-10	-20	-25	-30	-40	-50	-55	-65	-70	-80	-85	-95	-100	-110	-115	-125	-130	-140	
29 - 32	35	10	5	-5	-10	-20	-30	-35	-40	-50	-60	-65	-75	-80	-90	-100	-105	-115	-120	-130	-135	-145	
33 - 36	40	10	0	-5	-15	-20	-30	-35	-45	-55	-60	-70	-75	-85	-90	-100	-110	-115	-125	-130	-140	-150	
WINDS ABOVE 40 HAVE LITTLE ADDITIONAL EFFECT		LITTLE DANGER					INCREASING DANGER (Flesh may freeze within 1 minute)							GREAT DANGER (Flesh may freeze within 30 seconds)									
		DANGER OF FREEZING EXPOSED FLESH FOR PROPERLY CLOTHED PERSONS																					

ALCOM FORM 13b  
AUG 67

AAC - APO SEATTLE 98742  
Chart courtesy of Director of Plans and Training, USARAL



CASE No. 187  
THAWING BY EXCESSIVE HEAT



Fig. 1:  
*The extremity less than twenty-four hours after injury revealing the cyanotic, quite painful, and foul smelling hands, primarily lacking blebs, revealing the dusky changes of excessive heat applied to freezing injury. The patient, because of the severity of injury, had been brought to the operating room where controlled sympathectomy was performed.*



Fig. 2:  
*The tenth day following injury, revealing the unrelenting tissue demarcation and necrosis, with routine treatment still in effect, infection controlled by whirlpool and open treatment.*



Fig. 3:  
*At three weeks the digits are hard, rigid, soft tissue is completely mummified, absolute tissue death demonstrated. Again, there is evidence of infection, superficial only, at the area of tissue demarcation and amputation.*



Fig. 4:  
*Spontaneous amputation, bilateral, at the MP junction, at six weeks. The pattern is demonstrative of the hopelessness for recovery following the onslaught of gangrene when frozen tissues are cooked by excessive dry or wet heat.*



DEMONSTRATION OF REFREEZE  
(FREEZE-THAW-FREEZE-THAW SYNDROME)



Fig. 5:  
*Forty-eight hours post-thawing, in whirlpool bath. The distal areas and toes are dark, cyanotic, and demonstrate small proximal serosanguinous blebs. Pain quite marked at the demarcation line.*



Fig. 6:  
*Five days following thawing, the foot wet, edematous and insensitive; the forefoot markedly necrotic, demonstrating rapid demarcation and separation between injured and viable tissues.*



Fig. 7:  
*Seven days post-thawing, revealing rapid liquefaction necrosis of forefoot, separation of tissues with dissolution and liquefaction of ligaments, vessels, nerves, tendon and structures about the joint, the osseous structures held in place only by a skin envelope.*



Fig. 8:  
*This view, twelve days following thawing, demonstrates the irreversible tissue destruction common to freezing injury preceded by long immersion injury, or by thawing after a previous freezing. This appears to be a typical freeze-thaw=freeze-thaw pattern found frequently in mountaineers.*

SPONTANEOUS THAWING

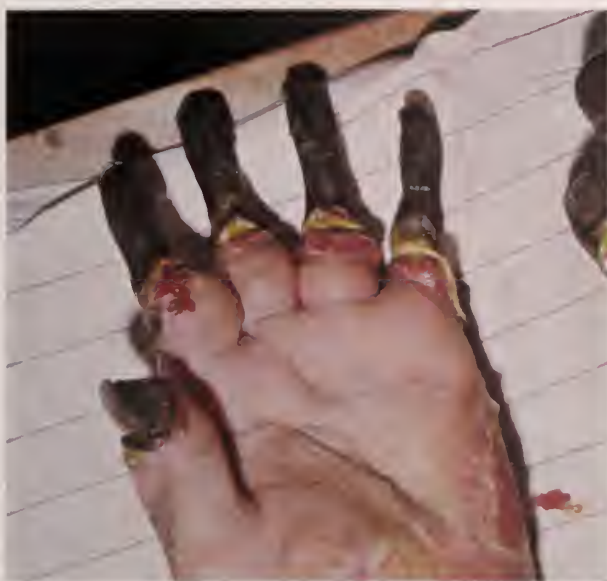


Fig. 9-10:

*Demarcation with obvious complete necrosis and spontaneous amputation is seen at six weeks. Demarcation was equal bilaterally, infection*

*superficial and minimal, silver nitrate lavage for comparison as a possible adjunct in controlling infection was utilized on the right.*



Fig. 11-12

*At three and a half months, he has adequate functional result for gross purpose, but poor anatomical result. Gross motion is permitted, the patient can feed and clothe himself, although with some difficulty, and has been discharged. He has, throughout the course, maintained an adequate and*

*rigid program of physiotherapy. At the end of the treatment there appeared to be no significant benefit from silver nitrate soaks as compared to the open granulating method, except that infection, if present, is more readily controlled utilizing 0.5%  $\text{AgNO}_3$  solution.*



RAPID THAWING IN WARM WATER (110° F.)



Fig. 17:

*A cold rigid forefoot without sensation or digital motion. Tissue compression and sock marks are obvious. Treatment was whirlpool bath and thawing at 110°F. for approximately twenty minutes.*



Fig. 18:

*Thawing was followed with an ominous burgundy hue. This has since been demonstrated in other cases, more often at temperatures greater than 110°F. The cyanosis remained for approximately six hours at which time small discrete blebs began to appear. Gross sensation was present after thawing and remained so until bleb development.*



Fig. 19:

*Over the next forty-eight hours large clear blebs developed ultimately extending to the digital tips. Failure of distal bleb formation, in the presence of proximal blebs, is an ominous prognostic sign.*



Fig. 20:

*Four months post-injury, the anatomy has been preserved, but the changes of deep injury are obvious, and include volar fat pad loss, subcutaneous fat loss, early IP joint contracture, nail changes, hypesthesia and hyperhidrosis. Epithelialization is complete. At the end of one year the extremity had adequate sensation, there was mild subcutaneous loss and interphalangeal contracture, with a few interphalangeal subarticular lesions present on X-ray examination. Increased sweating was present.*

FREEZING INJURY AND EARLY SYMPATHECTOMY



**Fig. 24:**  
*Bilateral equal injury, 36 hours post-thawing by rapid rewarming in warm water. Patient complained of severe pain, bilateral, and agreed to a control sympathectomy on the left.*



**Fig. 25:**  
*Nine days post-lumbar sympathectomy. Note absence of superficial infection as compared to the right, or control, side. Tissues are dry and there is marked diminution of edema on the left. Pain is absent on the left, present on the right.*



**Fig. 26:**  
*There is continued loss of edema and pain on the left, but some early demarcation of the tips of toes 2 and 3. Anhydrosis on the left as compared to hyperhydrosis on the right.*



**Fig. 27:**  
*Here, same data as Fig. No. 26, but appearance of a similar injury is much different. There is still some superficial infection (*Pseudomonas*); continued edema, and pain. At the sixth post injury week, both feet were similar in appearance except that there had been greater tissue loss on the left (part of volar tips 2 and 3).*



CASE NO. 7  
DELAYED THAWING BY ICE AND SNOW



**Fig. 13:**  
*The feet are approximately five days post-freezing and forty-eight hours post thawing. Here a very poor prognostic sign is evident. The blebs are all proximal, and are dark. The toes and distal tissues are without blebs or blistering, and are dusky, edematous, painless and insensitive. Phalangeal amputation is generally unavoidable with this pattern and may be anticipated from the date of admission — as early as twenty-four hours post-thaw.*



**Fig. 14:**  
*The plantar aspect of the foot at the same period as Fig. 13 — the blebs are proximal and the plantar aspect of the foot without sensation. The pedal pulses are diminished but present.*



**Fig. 15:**  
*Three months post-thawing, with the superficial infection at the junction of viable and gangrenous tissues held in control by whirlpool baths and aseptic care permitting the self-demarcation of the tissues, so that maximum length of foot is gained. Amputation is considered from this point on, once the tissue edema has subsided and there is no further tissue retraction.*



**Fig. 16:**  
*Amputation at the distal metatarsal Level- the patient was back in the Arctic the following winter and has been a trapper and hunter there for the past six years.*

EXTREMITY FRACTURE FOLLOWED BY FREEZING  
AND SPONTANEOUS THAWING



Fig. 21:

*Here the extremity demonstrates the cyanotic hue of the ischemic limb demonstrating vascular insufficiency. The pedal pulses are absent and the sensation is absent to the level of the malleoli. There are no blebs, proximal or distal, at this stage. This is a typical picture of extremity fracture or dislocation, thawed by other than rapid rewarming.*



Fig. 22:

*On the fourteenth day there was only minimal bleb formation and these dark and serosanguinous. The foot was without sensation and the digits were obviously gangrenous.*



Fig. 23:

*At the end of the third week after thawing, there was dry gangrenous change of most of the foot and the plantar pad. Tissue necrosis continued and the extremity was amputated at the level of the fracture.*



# SUMMARY OF TREATMENT OF THE COLD INJURED PATIENT

William J. Mills, Jr, M.D.

Confronted with the victim of cold injury, first consideration is a determination of the condition of the patient, as in any emergency. The degree of hypothermia present is of prime concern. General body cooling, and loss of heat, with exhaustion of caloric reserve and severe depression of core temperature may lead to death. Homothermic control is unstable and often lost to temperatures below 94°F. Continued cooling, unrelieved heat loss, may result in coma, and eventually cardiorespiratory failure even before the level of 88°F.

From resuscitation techniques perfected and utilized when general hypothermia is used as an adjunct to surgical anesthesia, we recommend rapid thawing of the patient with warm packs and blankets, or rapid thawing in a warm tub, or the use of warming fluids, or dialysis as mentioned in other parts of this issue of *Alaska Medicine*.

Whatever method chosen, one must understand the problem to obtain the best results. The intravenous infusion of glucose and water, with all respiratory aids including intubation and oxygen if necessary, are utilized, and preparation made for cardiac defibrillation, or intubation, or tracheostomy if necessary. Pre-existing anoxia, generalized, or of local tissues, and trauma with excessive blood loss, as well as alcoholic or drug stupor, may complicate treatment and recovery.

Thawing in a warm water bath, 90° to 106° F., (32 to 37.7° C.) will bring your patient rapidly to a responsive state. The change is dramatic, the patient becoming quickly alert, rational, and vocal. So rapid however, is this method of thawing, that the tissue liberation of acid end products of metabolism, and the sudden end result of metabolic acidosis may result in death by ventricular fibrillation in less than one to three hours after this mode of thawing. The result (death) may be the same by delayed thawing (that is by room temperature) without adequate supervision, death then often occurring in 24 to 48 hours.

Initial care, then, is directed to avoiding acidosis and its subsequent changes. Electrolyte monitoring by pH determination, pCO<sub>2</sub>, and pO<sub>2</sub> should begin immediately, and should be checked constantly as fluids are given, particularly after the use of sodium bicarbonate or THAM. Electrocardiographic monitoring should be performed. Venous blood is sufficient for pH readings. Due to rapid changes in electrolytes,

results should be obtained quickly. Occasionally, due to loss of electrolytes from gastric suction, or from the effect of the treatment of a stress ulcer, alkalosis may occur.

In order of prognosis, from best to worst, methods of thawing are (1) rapid rewarming in water (100 to 112°F. 37.7 to 44.5°C.), (2) gradual thawing at room temperature (the problem here is the variable room temperature between that of an average heated home to that of a cool cabin in the wilderness) (3) delayed thawing or thawing with ice and snow techniques (4) thawing by excessive heat (120° F. or higher). At present, rapid rewarming is favored, this method seeming to demonstrate the greatest tissue preservation and the most adequate early function especially in deep injury. Results by gradual thawing vary in deep injury, but seem satisfactory in the superficial injury patients. Ice and snow thawing gives variable results; most often poor, with marked loss of tissue. The use of excessive heat as a thawing method has resulted in disaster in most cases, especially with dry heat at temperatures of 150 to 180° F. (66 to 82° C.) (as the use of diesel exhaust, wood fire, stove heat).

Treatment generally can be directed into two categories. (A) *Before Thawing*. Here the *frozen part must be protected to avoid trauma*, should be *thawed in a whirlpool bath or tub water bath* or if nothing else is available, with warm wet packs at 100 to 112° F. (37.7 to 44.5° C.). *Temperatures should not exceed 112° F. or 44.5° C. The thawing is completed when the distal tip of the thawed part flushes. Sedatives or analgesics may be utilized if the thawing process is painful and cannot be tolerated. The part should not be massaged. Do not use rapid rewarming if the part has previously thawed.*

(B) *After Thawing*. When injury is severe, and deep, and hospitalization is required, *the extremities are kept on sterile sheets, with cradles over the frostbitten extremity to avoid trauma and pressure*. This is not necessary for upper extremities that may be laid out upon sterile sheets over the chest and trunk. *Treatment is open*, not occlusive, without the use of wet dressings, unguents, ointments, or petrolatum gauze. *Whirlpool baths are utilized twice daily for 20 minutes at a time, at temperatures between 90 to 95° F. Surgical soaps such as hexachlorophene or*

*betadine are utilized in the whirlpool. Occasionally after Moyer's method for burns, 0.5 per cent silver nitrate may be lavaged over the area of frostbite. The end result is similar to that of the soaps, hexachlorophene and betadine, epithelialization is similar, with one outstanding difference. Pain is less and infection, even superficial, is much less obvious using the silver nitrate solution. By the use of whirlpool, the debris is cleansed from the part, and superficial bacteria removed. The tissues are debrided without trauma when they are physiologically prepared to separate from the overlying eschar.*

*Generally blebs are left intact since the contents are sterile, as are the underlying tissues. The blebs are debrided or trimmed only if infected and contain purulent material. Escharotomy should be performed on the dorsum or dorsum or lateral aspect of the digits when the eschar is dry and has firmed sufficiently to have a cast effect on the digits limiting their joint motion. They will be debrided further in the whirlpool without prematurely exposing underlying granulation tissues. Unnecessary debridement or amputation should be delayed until sufficient time (often 30 to 90 days) elapses to demonstrate mummification and tissue death with no danger of further retraction of tissues.*

In recent years, the use of felt liners has been popular as a snow boot. Occasionally an extremity (s) immersed in overflow water or wetted by other means, has sustained freezing injury, and the felt liner wet, then hardens and constricts, acting as a tourniquet about the lower extremity.

If the extremity has remained in a frozen state for some considerable time, even rapid thawing and general supportive care will not be effective in restoring the circulation and a condition similar to anterior tibial compartment syndrome may be demonstrated clinically. *This condition may require fasciotomy.* This condition can be determined either clinically, by the use of arteriography, or injection of isotopes such as technetium 99m. The use of split thickness skin for large granulating areas or areas where skin cover is considered proper may have skin applied from the third to the fourteenth day. The results of skin graft are best following thawing by rapid rewarming. The pedicle grafting of full thickness skin is a late procedure.

*The use of antibiotics is not necessary except in deep infection. Cotton pledgets between digits will prevent maceration of tissues. Bedside digital exercises of all the joints are recommended, this done throughout the entire waking day, and Buerger's exercises for lower extremities are recommended 4 times daily at least. Narcotics*

generally are not utilized in the uncomplicated cases after initial thawing. Tranquilizers or aspirin will suffice for pain. In the very early stage, *sympathetic blockade, sympathectomy, anticoagulants, vasodilators, alcohol, and enzymes* have not proved particularly effective.

In patients with apparently equal bilateral injury, however, results of sympathectomy within the first 24 to 48 hours have demonstrated that, while there is no further preservation of tissue, there is (1) decrease in pain, (2) marked decrease in edema, (3) much less infection superficially or deep, and (4) or early and more proximal tissue demarcation.

Patients are kept in a pleasant environment, not relegated to corners of the hospital because of odor, or tissue necrosis. *The diet is high protein and high caloric, with vitamin supplements as needed and of your choice. When considered necessary, antitetanus therapy is recommended, particularly toxoid booster for those previously immunized. If for any cause, amputation must be performed, a modified guillotine procedure at the lowest level is recommended with secondary closure to be done at a later date. Dislocations and fractures pose interesting problems, and the dislocation particularly should be reduced immediately after thawing. The use of traction or trauma or manipulation or open procedures are done seldom and only then very carefully in the patient who had extremity fracture prior to his freezing. The fracture treatment should be conservative until the post thaw edema is eliminated. It may be that well padded plastic mold is the best method of treatment until there is cessation of edema. If open reduction of fractures or dislocations is required, great care must be utilized to avoid further vascular injury. Postoperatively, the operated part in a plastic posterior mold may still undergo whirlpool therapy and active digital exercises. The prognosis of this combined injury is poor because of injury to the regional vascular supply from fracture trauma, and then the added insult of superimposed freezing injury. It is here that fasciotomy may be required to relieve the deep structure pressures. Fluids are encouraged, dehydration is to be avoided and electrolyte balance maintained. Smoking is discouraged; alcohol may be permitted.*

The above is a basic program to which you may add any other therapy of choice. The following illustrations provide a visual summary of the salient factors in therapy.

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## SUMMARY



*Thaw rapidly in warm water (100 - 112°F.)*



*Avoid bleb rupture when transporting patient*



*Protect injured parts during bleb stage*



*Avoid premature debridement or surgical intervention*



*Do incise constricting eschar with lateral or dorsal slits to permit IP joint movement*



*Twice daily whirlpool bath will permit physiological separation of necrotic tissues from newly formed epithelium below*



*Constant MP and IP joint motion is demanded, from completion of thawing to end of treatment*



*Thaw the injured extremity by the rapid rewarming method and do an immediate reduction of the fracture or dislocation. Avoid further freezing constriction by cast or excessive traction. Avoid all neurovascular trauma. (The above injury demonstrates the result of an unreduced fracture-dislocation of the tarsal navicular and spontaneous thawing.)*



# ACCIDENTAL HYPOTHERMIA: MANAGEMENT APPROACH

William J. Mills, Jr., M.D.

It is apparent that much controversy exists regarding the most effective and the least hazardous method of warming the victim of accidental hypothermia here in Alaska and elsewhere in this country.

This short review is aimed at an immediate review of the problem and an effort to demonstrate a basic fundamental methodology to handle rewarming.

The elements of controversy in warming methods include "peripheral", as compared to "core rewarming", the development of reported phenomenon of afterdrop and its significance, and the etiology of cardiac irregularities, attributed to certain warming methods.

Recently an evaluation has been made of over fifty Alaskan case histories. The temperature ranges, measured by both low reading and standard clinical thermometers, varied from 96° F. (36° C.) to 70° F. (23° C.). A large number of these patients had temperature recordings of 94° F. when they actually were in a much colder state, and brought forth again the fact that many Alaskan hospitals still apparently do not have available low reading core temperature thermometers. The age of the patients studied varied from 24 hours old to 90 years of age. These victims were warmed by six methods. These included:

1. Uncontrolled spontaneous warming K-Thermia pad, Warm Blankets, Dry Heat
2. Spontaneous Warming, Controlled
3. Rapid Re-warming, Warm Water Bath or Whirlpool, External Wet Heat, Uncontrolled
4. Rapid Rewarming, Warm Water Bath or Whirlpool, External Wet Heat, Controlled, 90° - 106° F. (32° - 42° C.)
5. Peritoneal Dialysis, Controlled
6. Gradual Warming with Warm Fluids, I.V., and the Use of Anesthetic Breathing Apparatus in Surgery.

Of all this group, there were four deaths. In two cases the warming method did not include full physical and chemical body monitoring or control, and treatment of

the underlying abnormalities of the hypothermic state. These two were the first two patients treated in 1964 when interest in developing an adequate treatment method for hypothermia was begun by us. The other deaths are apparently the result of unresolved cardiac failure.

Most of the patients studied were dehydrated, demonstrated mild to severe acidosis and demonstrated evidence of hyperkalemia either after or during warming. The degree of awareness and consciousness was correlated with the level of hypothermia in both adults and children, and it became obvious that cerebation, response and even shivering ability was present at much lower levels than originally considered likely. Inherent was the danger that without a low reading rectal thermometer and with a moderately responsive patient, one might overlook a diagnosis of hypothermia. It was further noted that the optimum solution to the problem of hypothermia in the emergency area was dependent upon the time permitted to solve the complex metabolic and cardiac and chemical changes as they appeared. More time was obviously given the treating area to care for the patient by utilizing slower, spontaneous warming methods - (three to eight hours), and less was given when the rapid warming methods were utilized - (thirty minutes to one and one half to two hours). Under controlled warming, good result was demonstrated by all methods utilized. However, the patient with associated freezing injury appeared to obtain better extremity anatomical and functional result when the rapid warming and thawing methods were utilized.

It is helpful then in approaching the patient who presents suddenly in a very obvious state of hypothermia, to delineate the problem. The problem is:

1. Lowered core temperature
2. Decreasing function of metabolic system
3. Dehydration

4. Loss of caloric reserve
5. Enzyme system dysfunction
6. Hypoxia of tissues and transfer to anerobic metabolism
7. Metabolic acidosis
8. Renal dysfunction
9. Increasing loss of neuro-regulation
10. Fluid shifts and electrolyte imbalance
11. Metabolic ice box
12. In the field - death from total systems cessation or after rescue - life or death, depending on gradual, orderly controlled reorganization of organ systems.

If found alive by rescue personnel, the victim may be essentially in a "metabolic icebox", in a mid-lethal state, so that further exposure will result in death from vital organ cooling and warming may, if uncontrolled, result in death because of uncorrected acidosis, and the sudden effect of released metabolites or increased serum potassium levels resulting in cardiac excitation or hypovolemic shock.

It should be noted that the cold heart is in a fragile state and that it is possible that aggressive manipulation using C.P.R. (cardiopulmonary resuscitation) or efforts to cardiovert a heart not metabolically prepared to accept such stimulation, may result in irreversible cardiac failure.

Knowing the problem then, what are we attempting to do? The solution should include:

1. Safely, under control, rewarm the "cooled", "cooling" body, and elevate the core temperature
2. Obtain total physiological control of the patient by:
  - a. Adequate airway control
  - b. Restoration of fluid electrolyte imbalance
  - c. Correction of dehydration
  - d. Correct the acidosis and alkalosis
  - e. Restore renal function
  - f. Develop adequate intravenous access (multiple), CVP Line
  - g. Properly monitor the heart, vital signs, fluid intake and output
  - h. Recognize and/or treat all of the conditions prohibiting immediate recovery

In order to do this, it is necessary that you have immediate control of the patient's rescue environment or warming environment and immediate organization of the rescue and treating personnel. Depending on the warming method, the patient may rapidly come to responsive state and therein lies his great danger. While in the field in his "metabolic icebox", the patient is for awhile at least, unless cooling continues, cold but often alive. His dangerous period and likelihood of metabolic and cardiopulmonary failure lies in warming area and in the hospital regions or rescue regions.

Therefore a planned approach to the problem is essential and it is important that physiological control be obtained as soon as possible. In this regard, the careful patient handling, the establishment of the airway, thorough evaluation of the patient and the early

monitoring of temperature, electrocardiogram and urinary output is essential. The control, as noted above, includes initiation as soon as possible of the I.V. leads, blood gases and electrolytes, and repetitive monitoring of these values.

The correction of the hypovolemia, utilizing glucose and water solutions or physiological saline, once baseline blood gases and electrolytes are obtained, is demanded and then with those baseline studies, sodium bicarbonate may be given for correction of acidosis and Mannitol or Lasix to aid in development of renal perfusion. Fluids given should be warmed to physiological levels. Do not assume that the patient is in acidosis unless absolutely necessary, because acid-base values may indicate that for other disease or injury or loss of H<sup>+</sup> Ion, because of gastric suction the patient may occasionally be in a state of alkalosis.

Once the patient is under total system control, warming by the method best suited to the emergency area or hospital facility is utilized. These include:

1. Warm by your most familiar, effective method which may include:
  - a. Spontaneous warming - controlled
  - b. External rapid warming (90 - 106° F.)
  - c. Peritoneal dialysis
  - d. Question both b. and c. above
  - e. Extra corporeal (heated circulation) blood, which includes:
    1. Cardio-Pulmonary Bypass
    2. Hemodialysis
    3. Other technology
  - f. Anesthetic breathing apparatus support with total control
  - g. Warm inspired air with total control
  - h. Thoracotomy: Lavage: Controlled
  - i. However you wish: But have total control, and total familiarity with the method

Again, once the patient is under total system control, warming by your own familiar method or even spontaneous slow warming is utilized. Whatever the method, the purpose of the treatment begun is to:

1. Restore a normal blood volume and overcome dehydration
2. Restore the acid-base balance
3. Restore a proper electrolyte balance and avoid a post-warming hyperkalemic state resulting in cardiac excitation
4. Encourage a normal renal flow
5. Avoid serious cardiac arrhythmias and arrest of the heart, and in this regard it is recommended that consideration be given to the fact that the cold heart, at very low temperatures, is not responsive to defibrillation procedures and electroshock, and this heart in fact may not be in the true cardiac arrest state of the pattern familiar to us in a normothermic state, but may represent truly the very delayed metabolic response to severe cooling. It does not seem logical to markedly stimulate by closed chest massage or electrostimulation a heart that is unable to re-



spond to that stimulation at the low temperatures.

With respiration controlled with an adequate airway, utilizing intubation when necessary, or utilizing warm inspired air, or the utilization of an adequate respirator with temperature and humidity control, further total control is completed.

At this stage, it would appear that the central large hospital areas, or outlying hospital facilities, need not be concerned that they have not available all of the multiple methods of warming patients recommended. It would appear that those patients who had spontaneous controlled warming of the hypothermic state, with or without rapid rewarming of the frozen extremities, with use of mechanical respirator, or anesthetic gas machine if necessary, may be utilizing the safest method of warming particularly since it allows satisfactory time for continuous correction of any imbalance, as the patient approaches a normothermic state.

A recent meeting of the International Hypothermia Conference and Workshop was held at the University of Rhode Island, January 23, 1980. It was agreed that pending further laboratory investigation and further clinical data evaluation, CPR would be recommended only where the patient was totally unresponsive, with no signs of life. De-fibrillation was not advised at temperatures below 85° F. Most Alaskan patients successfully resuscitated, alive, did not require either CPR or de-fibrillation.

That group also agreed that the use of warm moist inspired air was not in deep hypothermia, a form of primary rewarming, but instead had value in preventing further core temperature drop.

Again: Accidental Hypothermia

The Problem: A cold patient in a metabolic ice box: Alive?

The Purpose of Care: Bring patient to a physiological responsive state, under homeothermic control - living.

Consider: The higher the temperature of the warming methods, or thawing methods, the less time you have to direct, control, obtain and maintain the normal physiological state.

The Method: Rewarm only under total patient control.

It has been noted that of all the cases studied, 14 or 27.5% of the group were warmed spontaneously; using K-pads, warm blankets, heated inspired moist air and total system control, and all survived. 17 patients or 33.3% were warmed by rapid rewarming in a tub at 100° - 106° F. including 10 patients with associated severe frostbite of the extremities, that were simultaneously thawed, and all survived. Both of these methods are available in even the smallest of our outlying hospitals.

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# SUMMARY OF TREATMENT OF THE COLD INJURED PATIENT

## HYPOTHERMIA

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**The Initial Management of Thermal Injuries  
The University of Alaska  
and  
Providence Hospital  
Anchorage, Alaska  
March 11, 12, 13, 1983**



## GENERAL HYPOTHERMIA

Confronted with the victim of cold injury, first consideration is a determination of the condition of the patient, as in any emergency. The degree of hypothermia present is of prime concern. General body cooling and loss of heat, with exhaustion of caloric reserve and severe depression of core temperature, may lead to death. Homothermic control is unstable and often lost at temperatures below 94°F. (34.5°C.). Continued cooling, unrelieved heat loss, may result in coma and eventually cardiorespiratory failure. Doolittle<sup>20</sup> has succinctly and precisely defined one major (and early) facet of hypothermia management—"In the very beginning, when concerned with the victim(s) of hypothermia, THINK HEAT."

Many hospitals and rescue units do not have thermometers or temperature probes that read below 94°F. (34.5°C.). This often delays or prohibits the proper diagnosis.

Many hypothermic patients are dehydrated and hypovolemic and demonstrate mild to severe acidosis and evidence of hyperkalemia either after or during warming. The degree of awareness and consciousness has been correlated with the level of hypothermia in both adults and children, and it is obvious that cerebation, physiological responses, and even shivering ability are present at much lower levels than previously considered likely. The optimum solution to the problem of hypothermia in the emergency area is dependent upon the time permitted to solve the complex metabolic and cardiac and chemical changes as they appear. More time is obviously given the treating area to care for the patient by utilizing slower, spontaneous warming methods — (three to eight hours) — and less is given when the rapid warming methods are utilized — (thirty minutes to one and one half to two hours). Under controlled warming, good result is demonstrated by all methods utilized. However, the patient with associated freezing injury appeared to obtain better extremity anatomical and functional result when the rapid warming and thawing methods were utilized in warm water, 95°-100°F. (35°-37.7°C.).

The problem presenting in hypothermia is:

1. Lowered core temperature
2. Decreasing function of metabolic system
3. Dehydration
4. Loss of caloric reserve
5. Enzyme system dysfunction
6. Hypoxia of tissues and transfer to anerobic metabolism
7. Metabolic acidosis
8. Renal dysfunction
9. Increasing loss of neuro-regulation
10. Fluid shifts and electrolyte imbalance
11. Metabolic ice box

12. In the field — death from total systems cessation; or after rescue — life or death, depending on gradual, orderly controlled reorganization of organ systems.

If found alive by rescue personnel, the victim may be essentially in a "metabolic icebox", in a mid-lethal state, so that further exposure will result in death from vital organ cooling and warming may, if uncontrolled, result in death because of 1) uncorrected acidosis, and the sudden effect of released metabolites or 2) increased serum potassium levels resulting in cardiac excitation, or 3) hypovolemic shock.

It should be noted that the cold heart is in a fragile state and that it is possible that aggressive manipulation using C.P.R. (cardiopulmonary resuscitation) or efforts to cardiovert a heart not metabolically prepared to accept such stimulation, may result in irreversible cardiac failure.

Knowing the problem then, what are we attempting to do? The solution should include:

1. Safely, under control, rewarm the "cooled", "cooling" body, and elevate the core temperature
2. Obtain total physiological control of the patient by:
  - a. Adequate airway control
  - b. Restoration of fluid electrolyte imbalance
  - c. Correction of dehydration
  - d. Correct the acidosis or alkalosis
  - e. Restore renal function
  - f. Develop adequate intravenous access (multiple), CVP Line
  - g. Properly monitor the heart, vital organs, fluid intake and output
  - h. Recognize and/or treat all of the conditions prohibiting immediate recovery.

In order to do this, it is necessary that you have immediate control of the patient's rescue environment or warming environment and immediate organization of the rescue and treating personnel. Depending on the warming method, the patient may rapidly come to responsive state and therein lies his great danger. While in the field in his "metabolic ice box," the patient is for awhile at least, unless cooling continues, cold but often alive. His dangerous period and likelihood of metabolic and cardiopulmonary failure lies in the warming area and in the hospital regions or rescue regions.

Therefore a planned approach to the problem is essential, and it is important that physiological control be obtained as soon as possible. In this regard, the careful patient handling, the establishment of the airway, thorough evaluation of the patient and the early monitoring of temperature, electrocardiogram and urinary output is essential. The control, as noted above, includes initiation as soon as possible of the I.V. leads, blood gases and electrolytes, and repetitive monitoring of these values.

The correction of the hypovolemia, utilizing glucose and water solutions or physiological saline, once baseline blood gases and electrolytes are obtained, is demanded, and then with those baseline studies, sodium bicarbonate may be given for correction of acidosis and Mannitol or Lasix to aid in development of renal perfusion. Fluids given should be warmed to physiological levels. Do not assume that the patient is in acidosis unless absolutely necessary, because acid-base values may indicate that for other disease or injury or loss of H<sup>+</sup> Ion, because of gastric suction the patient may occasionally be in a state of alkalosis.

Once the patient is *under total system control*, warming by the method best suited to the emergency area or hospital facility is utilized. These include:

First, warming by your most familiar effective method, as

- A. *External Passive Warming* to include dry clothing and dry blankets, insulated mats and warm shelter if in the field.
- B. *External Active Warming* as warm blankets, Norwegian charcoal body warmer, warming cradles, radiant heat, circulating warm water blanket, rapid rewarming in a tub, whirlpool or Hubbard tank.
- C. *Internal (Intracorporeal) Warming* as warm enemas, gastric lavage, inhalation warm moist oxygen or air, (100-110°F.) (37.7-43.3°C.) or warmed intravenous solution, (100-106°F.) (37.7°-41.1°C.)
- D. *Internal (Extracorporeal) Warming* including peritoneal dialysis, hemodialysis, veno-arterial shunt with extracorporeal heat exchange and disposable oxygenator and partial-cardio-pulmonary bypass.

The purpose of the treatment is to:

1. Restore a normal blood volume and overcome dehydration
2. Restore the acid-base balance
3. Restore a proper electrolyte balance and avoid a post-warming hyperkalemic state resulting in cardiac excitation
4. Encourage a normal renal flow
5. Avoid serious cardiac arrhythmias and arrest of the heart; and in this regard, it is recommended that consideration be given to the fact that the cold heart, at very low temperatures, is not responsive to defibrillation procedures and electroshock, and this heart in fact may not be in the true cardiac arrest state of the pattern familiar to us in a normothermic state, but may represent truly the very delayed metabolic response to severe cooling. It does not seem logical to stimulate by closed chest massage or electrostimulation a heart that is unable to respond to that stimulation at such low temperatures. This matter is currently the subject of much debate and investigation.

At this stage, it would appear that the smaller, outlying hospitals need not be concerned that all of the warming methods mentioned are not available to them. Those patients who had spontaneous controlled warming of the hypothermic state, with or without rapid rewarming of the frozen extremities, with use of a mechanical respirator (or anesthetic gas machine if available) most likely are utilizing the safest method of recovery. This is particularly so, since under *physiological controlled warming*, there is sufficient time for continuous correction of any imbalance, as the patient approaches a normothermic state.

## SUMMARY

A review of the literature, and the experience in Alaska, would seem to demonstrate that regardless of warming method, good results are had if the management included delineation of the immediate problem, immediate control of the patient's rescue environment, a thorough but rapid physical examination, and evaluation of the physiological state of the victim. Once under 'total physiological control' (monitoring heart, blood gases, electrolytes, and airway, with thorough evaluation of the patient as in any emergency) then warming by the most effective, familiar method is begun. There is reason to believe that the method of warming and even the depth of hypothermia to the level of 70-75°F. (21.1°-23.8° C.) is no more important, if as much, than having total physiological control of the patient. Many methods now in controversy would demonstrate better results if prior to warming effort was made to have complete control of the blood gases, pH, electrolytes, associated injury, and to begin correction of all deficits, particularly dehydration and hypovolemia and renal impairment.

Warming by any of our numerous modalities is critical to restoration of normothermia and preservation of life. Prior to warming, while the patient is in the 'metabolic icebox', still alive, there is time before choosing the heating method, to place the patient under total physiological control. Once that is done, warming methods, simple or complex, may then only be a function of time, not a matter of life or death. Such an approach permits any area at any time to provide care for the victim of hypothermia regardless of the available warming modes.

It is considered essential then, that prior to choosing the method of heating (warming), that time must be allotted (and in the very cold victim, for awhile safe in a 'metabolic icebox state') to diligently search for a.) the cause of hypothermia, b.) and all underlying factors that might prohibit an adequate result, and further c.) to perform total physical, chemical and physiological analysis of the patient's condition.



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# **SUMMARY OF TREATMENT OF THE COLD INJURED PATIENT**

## **FROSTBITE**

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**The Initial Management of Thermal Injuries  
The University of Alaska  
and  
Providence Hospital  
Anchorage, Alaska  
March 11, 12, 13, 1983**



## FROSTBITE

Frostbite is true tissue freezing and occurs when there is sufficient heat loss in the local area to allow ice crystals to form in the extracellular spaces, and extract cellular water.

Freezing injury, as found in Alaska, occurs by the following mechanism:

1. True frostbite: Superficial or deep.
2. Mixed injury: Immersion injury, (wet cold injury) followed by freezing, usually disastrous and often quite painful.
3. Freezing, thawing by any means, with re-freezing; again generally disastrous in result, with total tissue destruction and early mummification of distal tissues occurring within five days.
4. Hypoxia, high altitude environment injury, often with dehydration of tissues, due to general body dehydration and hypovolemia with extremity freezing. (Prognosis poor, especially if associated with compartment pressure syndrome.)
5. Extremity compartment compression, from any cause, followed by freezing. (Very poor results if compartment pressures are not relieved by fasciotomy.)
6. Extremity fracture or dislocation and superimposed freezing. The results are poor if the fracture or the dislocation is left unreduced. Best results appear to follow rapid rewarming techniques.
7. Hypothermia, associated with superimposed freezing injury of extremities. Paramount importance is given the restoration of heat to the victim, under total physiological control and monitoring. Best results for freezing injury appear to be associated with tub rewarming of the hypothermia and simultaneous thawing in warm water of the frozen extremity. The danger here is the sudden release of metabolites and release of excess amounts of potassium from muscle degradation and injury that may cause cardioplegia. The immediate balance of electrolytes and restoration of normal pH levels is imperative. The very excellent method of rewarming with peritoneal dialysis may require almost simultaneous warming by other means of the frozen extremity.

Present knowledge would indicate that the pathophysiological changes occur in two stages. First are changes occurring in and induced by the freezing state, namely, 1. Structural damage by ice crystal growth, 2. Protein denaturation, 3. pH changes (inter- and extracellular), 4. Dehydration within cells, 5. Loss of protein bound water, 6. Rupture of cell membranes, 7. Abnormal cell wall permeability, 8. Destruction of essential enzymes, 9. Ultra-structural damage to capillaries, 10. Consistent mitochondrial damage in muscle cells. During the thaw and post thaw stage, the changes may

include 1. Circulatory stasis, 2. Corpuscular aggregation, 3. Venule obstruction, 4. Piling of red cells back to capillary bed, 5. Development of hyaline plugs in the vascular tree, 6. Marked tissue edema, 7. Anoxia-ischemia of tissues, 8. Increased compartment space pressure, 9. Capillary and peripheral vessel collapse with eventually, if the process is not reversed, 10. Thrombosis, ischemia, regional necrosis, and tissue death.

In order of prognosis, from best to worst, methods of thawing are:

1. Rapid rewarming in water (100 to 106°F. — 37.7 to 41.1°C.)
2. Gradual thawing at room temperature (the problem here is the variable room temperature between that of an average heated home to that of a cool cabin in the wilderness).
3. Delayed thawing or thawing with ice and snow techniques.
4. Thawing by excessive heat (120°F. or higher).

At present, rapid rewarming is favored, this method seeming to demonstrate the greatest tissue preservation and the most adequate early function especially in deep injury. Results by gradual thawing vary in deep injury, but seem satisfactory in the superficial injury patients. Ice and snow thawing gives variable results, most often poor, with marked loss of tissue. The use of excessive heat as a thawing method has resulted in disaster in most cases, especially with dry heat at temperatures of 150 to 180°F. (66 to 82°C.) (as the use of diesel exhaust, wood fire, stove heat).

Much controversy exists regarding thawing methods, both here in North America and in Europe. From over 800 cases of freezing injury and 80 cases of hypothermia, some conclusions may be drawn.

- A. Thawing by excessive heat or by ice and snow and friction massage techniques usually yields poor results.
- B. Spontaneous thawing permits variable results and those results are often determined by the depth of injury, the duration of freezing and the patient's activity during survival and rescue and thawing.
- C. Rapid rewarming by external means appears to provide better results, but without question does not always give protection from tissue loss, especially in deep or long duration injury.
- D. It has been stated that rapid rewarming by internal means (warm intravenous fluids or arterial line fluids) at temperatures of 100-160°F (37.1-41.14°C) is more physiological and may be a method of choice in dissolving ice crystals and restoring cellular hydration. Though this method appears most logical and is a new consideration on the horizon of care, it has, in fact, been a method of choice for over ten years, at least in this area and elsewhere, in the treatment of combined hypothermia and freezing injury, by adding heat and restoring fluid volume with the heated solutions. The

results are still no better than by rapid rewarming methods. In addition, the development of an arterial line, especially in the area of ankle and wrist, may cause local arterial spasm and further decrease digital perfusion. The ideal method is obviously not yet at hand (at least for thawing of the frozen part) but the tissue loss is less now than in the past decades, regardless of thawing method. Major above-knee or below-knee amputations or amputation at wrist or forearm level are much fewer in number.

Treatment generally can be divided into two categories:

- A. *Before Thawing.* Here the frozen part must be protected to avoid trauma. (Is there danger of irreversible injury at the frozen-nonfrozen interface, if motion occurs at that level, fragmenting partially frozen tissues?) should be thawed in a whirlpool bath or tub water bath or if nothing else is available, with warm wet packs at 100 to 106°F. (37.7 to 41.1°C.). At Providence Hospital in Anchorage, Alaska, we have since 1963 used a hoist (crane), electrically operated, with the patient lowered into a Hubbard Tub, with whirlpool, full body. This method has also been used for rewarming of the victim of hypothermia, or the warming-thawing of the combined injury, hypothermia, with extremity freezing. Temperatures should not exceed 106°F. or 41.1°C. The thawing is completed when the distal tip of the thawed part flushes. Sedatives or analgesics may be utilized if the thawing process is painful and cannot be tolerated. The part should not be massaged. Do not use rapid rewarming if the part has previously been thawed.
- B. *After Thawing.* When injury is severe and deep, and hospitalization is required, the extremities are kept on sterile sheets, with cradles over the frostbitten extremity to avoid trauma and pressure. This is not necessary for upper extremities that may be laid out upon sterile sheets over the chest and trunk. Treatment is open, not occlusive, without the use of wet dressings, unguents, ointments or petrolatum gauze. Whirlpool baths are utilized twice daily for 20 minutes at a time, at temperatures between 90 to 95°F. Surgical soaps such as hexachlorophene or betadine are utilized in the whirlpool. Occasionally after Moyer's method for burns, 0.5 percent silver nitrate may be lavaged over the area of frostbite. The end result is similar to that of the soaps, hexachlorophene and betadine, epithelialization is similar, with one outstanding difference. Pain is less and infection, even superficial, is much less obvious using the silver nitrate solution. By the use of whirlpool, the debris is cleansed from the part,

and superficial bacteria removed. The tissues are debrided without trauma by the whirlpool action, when they are physiologically prepared to separate viable tissue from the overlying eschar.

Recently one percent Silvadine solution (Silver Sulfadiazine) has been utilized on open wounds secondary to freezing injury when severe drying and premature blood rupture has occurred with apparent superficial infection. Its use occasionally prevents eschar separation apparently by inhibiting proteolytic enzyme bacterial growth.

Generally blebs are left intact since the contents are sterile, as are the underlying tissues. The blebs are debrided or trimmed only if infected and contain purulent material. *Escharotomy* should be performed on the dorsum or lateral aspect of the digits when the eschar is dry and has firmed sufficiently to have a cast effect on the digits, limiting their joint motion. Digits will be debrided further in the whirlpool without prematurely exposing underlying granulation tissues. *Debridement or amputation should be delayed* until sufficient time (often 30 to 90 days) elapses to demonstrate mummification and tissue death with no danger of further retraction of tissues.

In recent years, snow boots with felt liners have been popular. When the extremity(s) is immersed in water or the felt wetted by any means (melted snow) the felt liner may shrink, contract and freeze. Extremity freezing may follow, complicated first by vascular occlusion, the contracted felt liner acting as a tourniquet. Similarly, neoprene or rubber scuba boots used by the mountaineer may cause occlusion of circulation at high altitudes. This is because of pressure changes at lowered atmospheric pressure. Freezing, following this pre-existing vascular occlusion, terribly complicates the injury and final result.

If the extremity has remained in a frozen state for some considerable time, even rapid thawing and general supportive care may not be effective in restoring the circulation and a condition similar to anterior tibial compartment syndrome may be demonstrated clinically. *This problem may require fasciotomy.* This condition can be determined either clinically, or by measuring compartment pressures, by the use of arteriography, or injection of isotopes such as technetium 99m.

Isotope studies have been performed as a diagnostic aid of cellular perfusion for over ten years. Doppler ultrasound has been used as a vascular study tool. Interestingly, Thermal Unit patients at Providence Hospital, with evidence of good Doppler pulses in the distal extremities (distal digital vessels) have had conflicting isotope evidence of failure of extremity perfusion in the same area. In all cases but one, the isotope study was the accurate one. Obviously, large digital vessels may for a short while remain patent, even when the deep capillary system is blocked. Failure of sophisticated tools is demonstrated too in the use of devices to measure com-



partment pressure. If your clinical judgement and experience advises that an immediate fasciotomy is required, and the pressure transducer measuring device indicates that the pressure is high but not lethal, or indicates a marginal reading, then often it is better to trust your experience. A later measurement may indicate sudden pressure increase. A delay in performing the fasciotomy may be disastrous. This diagnostic problem may be avoided by the use of continuous pressure monitoring. The pitfalls are many. The monitoring device is still only a machine, and your studied concerned opinion to perform the fasciotomy may preserve the limb.

The use of split thickness skin for large granulating areas or areas where skin cover is considered proper may have skin applied from the third to the fourteenth day. The results of skin graft are best following thawing by rapid rewarming. The pedicle grafting of full thickness skin is a late procedure.

The use of a mesh skin graft at the time of fasciotomy or soon after, decreases the morbidity and lowers the incidence of scarring and infection.

*The use of antibiotics is not necessary except in deep infection. Cotton pledgets between digits will prevent maceration of tissues. Bedside digital exercises of all the joints are recommended, this done throughout the entire waking day, and Buerger's exercises for lower extremities are recommended four times daily. Narcotics generally are not utilized in the uncomplicated cases after initial thawing. Tranquilizers or aspirin will suffice for pain. In the past, sympathetic blockade, sympathectomy, anticoagulants, vasodilators, alcohol, and enzymes have not proved particularly effective.*

In the past, the use of Hyperbaric Oxygen chamber, single man unit, at two atmospheres of Oxygen appeared to be beneficial in post thaw frostbite therapy. Further evaluation of this adjunct method is planned as a part of the Department of High Latitude Study, University of Alaska, Anchorage.

In patients with apparently equal bilateral injury, however, results of sympathectomy within the first 24 to 48 hours have demonstrated that, while there is no further preservation of tissues, there is:

1. Decrease in pain
2. Marked decrease in edema
3. Much less infection, superficially or deep, and
4. Early and more proximal tissue demarcation

More recently, however, and still in process of evaluation, sympathectomy and vasodilators, and sympathetic blockade have been determined to be of good effect, following fasciotomy. I suspect the previous irregular results often reflected effort to perform effective sympathectomy when the problem may have included regional vascular compartment pressure block. Particularly effective has been the use of Phenoxybenzamine Hydrochloride (Dibenzylamine), given 10 mgm. daily and increased to 20 to 60 mgm. per day, depending upon effect and need. The drug is used for vaso spasm and

appears to be an effective alpha adrenergic blocking agent. It is important that the patient be well hydrated after surgical or chemical sympathectomy. Pain varies with each individual and with the type of injury, the degree of edema, and the presence or absence of infection. It is lessened by immediate physiotherapy, activity, and whirlpool bath. In severe cases of immersion injury, with edema, prior to fasciotomy, or with high level extremity freezing, post thaw, pain relief is provided with continuous epidural block, for 24 to 48 hours, repeated if necessary. This is especially effective if accompanied by fasciotomy in severe cases with associated increased tissue compartment pressure.

In the past four years, especially in the pre-injury patient with a labile vasomotor peripheral vessel response, biofeedback has been utilized to increase the hand and foot circulation. This has been utilized as well in the post-thaw extremity freezing victim.

New cultural patterns may establish changes in injury. For many years in Alaska freezing of ears, often with tissue loss, especially in children, primarily male, was not uncommon. The injury occurred in skiers, skaters and snowmobilers. With the advent of long neck length hairstyle, frostbite of the ear was seldom seen for almost a decade. With the return of the short hairstyle, the frozen ear pattern is with us again.

Similarly, the running shoe or tennis shoe foot style has permitted freezing. Even in Alaska, coastal or interior areas, regardless of the low temperature, wind or snow depth, Alaskan students (and many adults) risk freezing and nonfreezing injury in inadequate footwear. Their injury toll is almost matched by the cross country skier competing in low temperatures or backpacking in wilderness areas wearing ultralight low-cut shoes with toe clips.

Alcohol and drug abuse may contribute to hypothermia and freezing injury by impairing mental and physical function. Recently it has become apparent that the nasal "snorting" of cocaine, by causing constriction of mucous membranes and the nasal arterial supply, has allowed serious frostbite of the nose, an area usually so well provided with blood supply that deep injury was considered rare.

Patients are kept in a pleasant environment, not relegated to corners of the hospital because of odor, or tissue necrosis. *The diet is high protein and high caloric, with vitamin supplements as needed and of your choice. When considered necessary, antitetanus therapy is recommended, particularly toxoid booster for those previously immunized. If for any case, amputation must be performed, a modified guillotine procedure at the lower level is recommended with secondary closure to be done at a later date. Superficial or deep infection is often found in the extremity requiring guillotine amputation. Secondary closure after the amputation may be more successful when accompanied by closed suction-irrigation, the irrigation fluid (0.9% Sodium Chloride) flowing at 100 cc per hour, with a flush of 50 cc of an-*

tibiotic solution of your choice every hour. Dislocations and fractures pose interesting problems, and the *dislocation particularly should be reduced immediately* after thawing. *The use of traction or trauma or manipulation or open procedures are done seldom* and only then very carefully, in the patient who had extremity fracture prior to his freezing. *The fracture treatment should be conservative* until the post thaw edema is eliminated. It may be that a well-padded plastic mold is the best method of treatment until there is cessation of edema. If open reduction of fractures or dislocations is required, great care must be utilized to avoid further vascular injury. Postoperatively, the operated part in a plastic posterior mold may still undergo whirlpool therapy and active digital exercises. The prognosis of this combined injury is poor because of injury to the regional vascular supply from fracture trauma and then the added insult of superimposed freezing injury. *It is here also that fasciotomy may be required* to relieve the deep structure pressures. Fluids are encouraged, dehydration is to be avoided and electrolyte balance maintained. *Smoking is discouraged;* alcohol may be permitted.

The above is a basic program to which you may add any other therapy of choice including low molecular weight, Dextran, vasodilators, anticoagulants, hypotensive agents, sympatholytic drugs, and thrombolytic agents. Despite the best intended treatment, regardless of thawing method, indicated drugs or surgical care, some results are unexplained disasters. The poor result may be due to extended depth and duration of freezing, repetitive freeze-thaw-refreeze injury, underlying circulatory deficit or other cause. The post injury state may demonstrate 1. Freezing injury with post thaw vasoconstriction, 2. Freezing injury with arterial-venous-capillary thrombosis, and 3. Severe cellular destruction as a result of the freezing. Knowing the choices above will help choose the proper drug therapy. Anticoagulants (heparin), vasodilators (priscoline) or hypotensive adrenergic blocking agents (Guanethidine, Reserpine), including sympatholytic drugs (Dibenzylamine) may aid in the initial phase of care especially in the absence of deep thrombosis. The plasma volume expander, Low Molecular Weight Dextran, used early, is thought to prevent, diminish, or reverse red cell aggregation in the capillary tree. For deep occlusive thrombus formation, the use of thrombolytic enzymes, Streptokinase and Orokinase, are being evaluated. The risk of hemorrhage and lysis of fresh fibrin, may limit the use of these drugs with associated trauma, especially head injury where a Cerebral Vascular bleed may be of concern. The use of these drugs then may require special local and regional techniques. For the problem of severe or total cellular destruction, there is at the moment little help.

The cartilaginous structures in children, particularly the epiphyseal plates and non-ossified carpal and tarsal bodies are susceptible to cold insult and injury. Total necrosis of those organs is rapid and at present, regardless of methods of thawing and post thawing care, the injury is apparently irreversible.

It has become apparent that from a review of many cases, the following should be considered.

1. Do not attempt thawing where there is danger of refreezing the injured part.
2. There is probably tissue damage that occurs at the level of the nonfrozen-frozen interface in the process of survival, rescue or early extremity handing during treatment.
3. As one major aspect of etiology, it would appear that the onset of hypothermia and frostbite may be result of general overall dehydration and hypovolemia, resulting in further local distal tissue dehydration.
4. It, again, is noted that often the major disasters occur when the individual has often self-treated the extremities by an extreme of thawing temperature, using excessive heat as campfire, diesel exhaust or oven heat. When there is failure of adequate hydration following injury, results are poor. If there is failure to recognize increased compartment tissue space pressures and relieve them, arterial access to the injured part, and venous return is made difficult, if not impossible.
5. All the above problems may assume little importance when one considers that the thawing method is often out of the hand of rescue worker or attending physician since a large number of patients brought to Emergency Room areas or major hospital areas have already had thawing occur, either deliberately by their own methods or inadvertently in the process of awaiting rescue or during rescue procedures. This, then, makes most important the post-thawing techniques and care.

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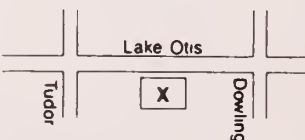
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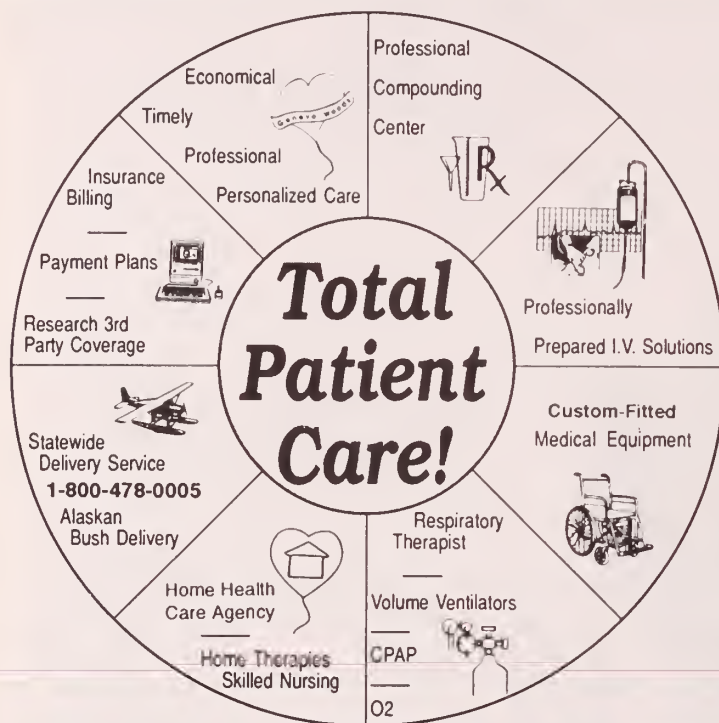


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# COMMENT AND RECAPITULATION

by William J. Mills, Jr., M.D.

The editor of *Alaska Medicine*, Donald R. Rogers, M.D. and his staff, by reprinting our previous papers from *Alaska Medicine*, has enabled me to review (under deadline) our original data, and so to reconsider the diagnosis and treatment of cold injury, particularly freezing. I am grateful to them, to be given an opportunity to present the development of the Alaska regimen of care of the frozen extremity.

Most obviously, much credit is due to the physicians throughout Alaska, and outside Alaska, too, who referred patients to our program. The large patient load, gained from all Alaska, seen in consultation or for primary or secondary care, allowed us to develop as the years passed, a system of care for the cold injured patient.

I am indebted also to Captain Richard Worthington, U.S. Navy Medical Officer in command of the Kodiak Naval Hospital in the late 1950s. Through his intervention and interest, a research grant was obtained from the office of Naval Medical Research to continue our study of cold injury. In 1980, a major grant from the Alaska State Legislature permitted the founding of the Center for High Latitude Health Research at the University of Alaska, Anchorage. That research program was best known for its activity on Mt. McKinley at the research stations at 7,300 and 14,300 feet; for the investigative studies during the Iditarod Race from Anchorage to Nome; and clinical activity at the Thermal Unit, Providence Hospital.

Because of the required support that was expertly rendered over the years, special thanks are given to the radiology and pathology departments at the Alaska Native Medical Center; Humana Hospital; Providence Hospital; and the U.S. Air Force Hospital, Elmendorf. And not least, this program must credit the medical, nursing and clerical staff of the Providence Hospital Thermal Unit, for they provided superlative suggestions for cold injury care, and diligent patient care, too, as well as needed consultation, and up-to-date physiological monitoring and interpretation of data.

I must add here (for should this comment escape the editorial ax, I may never again have such an opportunity) that as a physician, I have been often accused of "chauvinism" in regard to nurses and nursing -- and even as often, accused (justifiably?) of being a proponent of the "hand maiden" era (was it just yesterday?). So, I admit that I am duly impressed -- no, in awe -- of the "modern" nurses in the Thermal, Emergency and Intensive Care Units, who "in a blink of an eye" and "quick as a flash" manipulate banks of space-age, computerized recorders and equipment, and better yet, offer immediate translation. In this milieu of technology, the horse and buggy, model "T" and model "A" does are left scratching their heads. So, for many of us, only the art is left; the new age nurse has captured the science. Now, if she would only take night and E.R. calls. . .



Fig. 1.

Dialysis team nurse operating hemodialysis unit for hypothermia complicated by disseminated intravascular coagulation.



## Comments on this issue of *Alaska Medicine* -- From then (1960) until now (1993)



Fig. 2.

Mt. McKinley, the site of medical research camps at 7,300 and 14,000 feet. From April to July, multiple victims of mountain accident, hypothermia, freezing injury and altitude types illness are rescued here. Truly, with wide open unrehearsed protocol, Mt. McKinley is one of the worlds greatest clinical and physiological outdoor laboratories in the world. No institutional review board would permit scheduled experimentation of the type that weekly, or even daily, occurs on the slopes of this 'cold, arctic mountain.' It is often little appreciated, especially by European and Asian climbers, how dangerous the mountain is, with severe cold, raging storms and commonly occurring avalanches. This mountain, and its climbers, has given us an opportunity to study year after year, the effect of altitude, and cold.

Thirty-three years ago, Doctors William Mills and Robert Whaley reviewed 51 cases of freezing injury. They published their findings in the March 1960 issue of *Alaska Medicine*. They were joined in the December 1960 and the June 1961 issues by Dr. Winthrop Fish, who added to the previous developing system of care, the proposal that the use of a radioactive isotope of iodine ( $I^{125}$ ) be considered to aid in assessing blood flow in the cold injured extremity.



Fig. 3. C-43-60

Radio-isotope scan, 11-27-60, after method of Dr. Winthrop Fish, utilizing first  $I^{131}$ , then  $I^{125}$ . This and following procedures utilizing Technetium-99m has permitted us great accuracy in vascular perfusion determination, as well as prognosis, immediately after thawing.

It seems pertinent to remark here, that our journal *Alaska Medicine*, was born just a year before these articles on frostbite were published (B.D. March 1959). The journal then was neither peer reviewed, nor in the Index Medicus. Our interest in the early days, certainly was not peer review or any sort of Index recognition, but purely a problem of financial survival. In the beginning, the editors not only edited, but sold advertising, solicited papers from everywhere, especially from our own



Fig. 4.

Oscillating washing machines (here a Kenmore) were used in absence of whirlpools at the hospitals and homes before 1960.



**Fig. 5. C-79.**

**Frame #1:** Hard, cold, immobile frozen hands (bilateral) thawed in warm water (106 degrees F.) for less than 20 minutes.

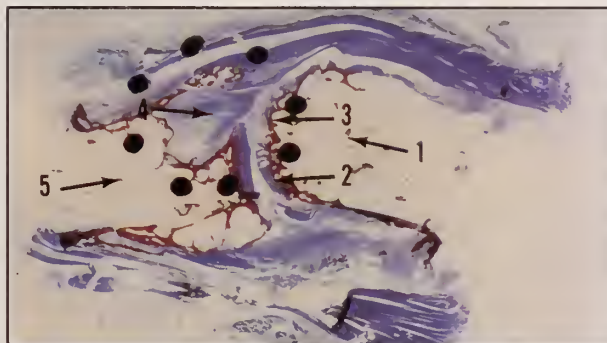
**Frame #2:** Because of second onset of cardiac episode, warming stopped prior to flushing of finger tips.

**Frame #3:** On 3-23-61 post thaw proximal blebs, with cyanotic tips.

**Frame #4:** Two months post thawing, gangrenous finger tips.

**Frame #5:** Mummification, separation of necrotic tissues.

**Frame #6:** Three and one half years after thawing, hands demonstrate flexion contractures of IP joints, nail deformity, intrinsic muscle atrophy.



**Fig. 6 C-79.**

1. Proximal phalanx mid finger right hand, site of post mortem biopsy 15 years post frostbite.
2. Degenerative articular cartilage.
3. Segmental destruction of subarticular bone P.I.P. joint.
4. Periosteal destructive lytic lesion of bone with invasion of joint surface by dense collagenous tissue.
5. Middle phalanx-trichrome stain.

**Fig. 7. C-10**

An elderly woodcutter, severe frostbite injury to feet and hands, with hypothermia. Rx tub warming. Referred for transmetatarsal amputation. Careful dissection of gangrenous epidermal cover revealed healthy epithelializing tissue under the eschar.







**Fig. 8 C-79.**  
AP right hand, date of freezing 3-23-61.  
No degenerative joint changes visible.



**Fig. 9 C-79.**  
AP, right hand with early evidence of small discrete, periarticular IP joint destructive changes. Osteoporosis and early cortical destruction in periarticular areas.



**Fig. 10. C-79.**  
Multiple areas of lytic lesions in IP joints, three one half years after freezing episode. Increased osteoporosis with marked narrowing of joint spaces, and evidence of cartilage destruction.

membership, and wrote papers as well, to keep the journal afloat. Our recognition, outside Alaska, was less than flaring.

This fact was brought home to me by a famed colleague in England, who at a meeting there noted in introduction that, "Dr. Mills and his associates in Alaska did good work, but published their material in a small, little known, obscure medical journal, not readily available to the rest of the world." I related this comment to my co-worker, Dr. Robert Whaley, explaining that I considered it our duty to publish our material in our own state journal (a truly provincial attitude, as a demonstration of home-town support). Dr. Whaley, in a more cynical, but candid comment said, "Actually, lest you forget, you were then editor-in-chief of *Alaska Medicine*, and I was an editor; so that by submitting the papers to *Alaska Medicine* for publication, we were assured of their immediate acceptance."

Our aim in the first series of papers (1960-61) was as follows:

1. To demonstrate the efficacy of rapid rewarming, in a tub or whirlpool bath. (It is of interest that in 1955, Providence Hospital in Anchorage had no physiotherapy department or whirlpool bath; so that our first patients requiring whirlpool therapy sat on a high wooden stool, placing cold injured extremities in an oscillating Kenmore washing machine.) This is less frightening than the commercial fisherman, victim of freezing, who utilized an outboard motor in his bathtub in lieu of a whirlpool.
2. To establish a rewarming temperature of 42-47 degrees C. (110-118 degrees F.), listed in

experimental literature as probably the most effective rewarming temperature. (It was not long before this temperature was deemed to be too warm and was dropped to 37-41 degrees C. (100-106 degrees F.).)

3. To avoid further trauma to the frozen or thawed part (from excess heat, cold or mechanical trauma).
4. To especially avoid refreezing of the thawed extremity, in rescue care or transport.
5. To recognize and treat associated trauma, recognizing the need to immediately care for the condition often present, with frostbite, hypothermia. And to care for the presenting problems there of hypovolemia, dehydration, electrolyte imbalance, and acidosis.
6. To utilize whirlpool twice daily in the post-thawed stage, at average skin temperatures of 32-34 degrees C. (90-93 degrees F.). This bath, given for 20-30 minutes twice daily, provided massage and stimulation to involved tissues, and washed off superficial bacteria accumulating on the surface of vascular deprived tissues, reducing the bacterial count on a regular basis.
7. To avoid infection by the above methods, by clean and sterile bedside techniques, with immediate culture of any area evidencing bacterial invasion.
8. To avoid, unless absolutely necessary, early debridement or amputation. (The whirlpool bath usually is effective in separating and debriding necrotic tissues in an atraumatic fashion.)
9. To encourage near constant digital exercises, utilizing a bedside program of physiotherapy to encourage range of motion (from initial thawing) of the digital joints.

10. Initially, we utilized ultrasonic therapy post-thawing. It soon became apparent that this was a dangerous tool. Ultrasound was quite effective in superficial injury, but in deep injury appeared to be a harmful modality, increasing tissue necrosis, particularly in the absence of an adequate vascular supply. The use of ultrasound or any deep diathermy was dropped from the regimen of care.
11. To simplify the existing diagnosis of depth of injury. It became apparent that degree, depth or severity of injury, described as first degree, second degree, third degree, fourth degree, was a diagnosis more suited to the pathologist looking with a microscope at debrided or amputated tissue. For the clinician, the diagnosis or description of superficial or deep injury was a more satisfactory statement of the frozen and post-thawed extremity in the early days of treatment, given the tools at that time and given the fact that once the superficial tissues were frozen, involvement of tissues below that was difficult to ascertain.
12. To determine prognosis by clinical appearance and examination. A good prognosis was expected if the blisters or blebs were clear or pink, and extended to the digital distal tips. A poor prognosis was anticipated if the blebs were dark or hemorrhagic and remained proximal on the extremity above the MP joints. The absence of blebs, with marked cyanosis and coolness, pulseless as in a freeze-thaw-refreeze injury were, of course, the poorest prognostic signs of all.
13. It was recommended that blebs and bullae remain closed, since the contents of blebs were usually sterile. In the event of bleb rupture, evidence of infection, or incision to drain purulent material, the exposed tissues were routinely cleansed twice daily in the whirlpool bath; the bath containing also a bacteriostatic-bacteriocidal solution such as hexachlorophene -- 4 percent chlorhexidine gluconate, and povidone-iodine. The choice of surgical soap is yours; the purpose is to reduce bacterial colonization.

So developed, as seen in the 1960-1961 papers, an Alaskan system of care -- a regimen with the initial concept utilizing proper bedside care and techniques, medical care as required and surgery, also when required. The concept included the addition of new treatment methods as they appeared in the literature, or surfaced as we continued our research and care. Thus, as new methods, drugs, or acceptable techniques appeared on the horizon; we were able to add to, discard, or alter our own previous principles of care.

Over the years, this initial regimen of care and the basic core concept that developed has remained

fundamentally unaltered, and has many times over, still re-emphasized five important tenets, namely:

1. To avoid refreezing the thawed extremity, at all costs.
2. To utilize rapid rewarming in warm water, preferably in a whirlpool bath. The preferred temperature range, altered several times, of the thawing water may vary from 32 degrees C. (90 degrees F.) to 41 degrees C. (106 degrees F.). An adequate range may be considered to be from 32 degrees C. to 38 degrees C. (100 degrees F.). The higher ranges of thawing are more painful and often border on the edge of excessive heat. In fact, only a few more minutes are required at the lower ranges (32-38 degrees C.) to allow thawing of frozen tissue and demonstrate distal digital flushing, the sign of returning circulation.
3. To avoid infection and accumulation of superficial bacteria by twice daily whirlpool baths in a tub, utilizing the surgical soaps previously listed, Hibiclens, Betadine, Phisohex. The water temperature after the initial thawing and in the daily baths should not exceed 32 degrees C. (90 degrees F.).
4. To pursue immediate post-thaw restoration of joint motion (any joint at any level of involved, thawed tissue).
5. To examine early, and often, for evidence of compartment pressure syndrome, and to perform early escharotomy and fasciotomy, if indicated.

In 1973, a further report of 200 cases was made. In the interim from 1960 to 1973, the Alaska regimen had developed to include routine use of technetium 99<sup>m</sup> to determine cellular perfusion and tissue viability, and to aid in diagnosis and prognosis. The use of escharotomy and fasciotomy became a standard care procedure when required.

It became more apparent that severe injury, with or without rapid rewarming, often included signs of increased compartment pressure. Various methods of measuring compartment pressure have been utilized by us in the past, but the Stryker stick apparatus (or the 295 intra-compartmental pressure monitor system) is favored at the present time.

Experience has dictated that although capillary pressures greater than 37-40 mm. of Hg are suspect, one must consider fasciotomy in the presence of clinical evidence of vascular compromise. The ultimate decision to do the fasciotomy, however, rests with the attending physician or surgeon, and that judgment often may demand compartmental release regardless of pressure readings. This is important to note since unless the compartment pressure reading is on continuous



**Fig. 11.** Case #731-81

**Frame #1:** The patient, a mountain pilot, was injured when his plane crashed on the ridge between Peters and Kahiltna Glaciers. He sustained freeze-thaw-refreeze injury of hands and feet, fractured multiple ribs, compression fracture of L5, and fracture of the first through fourth transverse processes. Exposure was five days. Of the four occupants of the plane, the pilot and one passenger survived. The patient lost his boots in an avalanche, and all survival gear in the plane was covered with snow and impacted in the fuselage after the avalanche. The temperature was low, typical of Mt. McKinley, with severe gusting winds.

**Frame #2:** After climbing party and helicopter rescue, the patient presented in the emergency room with feet and hands frozen, the feet frozen to the malleolar level and stockings encased in ice.

**Frame #3:** Removal of stockings demonstrated ice-covered feet, hard, cold and immobile clear to the level of the ankle or the malleoli bilaterally.

**Frame #4:** The technetium scan the day after admission revealed absent perfusion of both feet and total lack of capillary perfusion almost at the malleolar level.

**Frame #5:** Prior to thawing, the feet demonstrate severe freezing to the malleolar level, without sensation or motion, rock hard, yellow, bordering on pale gray, red and mottled, cold with ice crystals demonstrated still in the skin and the foot pulseless.



**Fig. 12.** Case #731-81

**Frame #1:** Following rapid rewarming of the hands and feet, marked tissue swelling occurred. After the demonstration of vascular occlusion by technetium scan, bilateral fasciotomy was performed. Some fascial tissue pressures had demonstrated pressure levels exceeding 75 to 80 mmHg bilaterally. Fasciotomy included neuromuscular decompression of foot and ankle. These tissues at surgery revealed severe swelling, with the arterial system pulseless. Blood flow increased post-fascial release, even immediately after release while still on the operating table.

**Frame #2:** Despite the fasciotomy, necrosis and gangrenous changes continued to the malleolar level.

**Frame #3:** Five weeks post-thawing, is demonstrated the previous guillotine amputation of the right foot at the malleolar level.

**Frame #4:** Amputation revision and secondary closure, with a drain, is illustrated. In many cases, this procedure is performed using closed suction irrigation - all to avoid persistent wound infection and to dilute bacteria localized in the wound.

**Frame #5:** After amputation stumps are granulating well, split thickness skin graft is applied.

**Frame #6:** Fully covered, nondraining amputation stumps four month post-injury, are ready for stump revision. (See frame #4).





**Fig. 13. C-101**  
Freezing injury, followed by thawing by excessive heat (hot oven) results in rapid mumification and dry gangrenous tissues with early demarcation, often by the fifth day.



**Fig. 14.**  
The cardinal danger in survival and rescue, the freeze-thaw-refreeze injury.

**Fig. 15. C-87-61.**

**Frame #1:** A 23-year-old dog team driver who lost dogs on the trail and froze both hands. Ambient temperature -14 degrees C., wind 10 miles per hour, exposure time one to two hours. Delayed warming with snow and ice water was followed later by warming in warm water. Note the clear, pink, large distal blebs, except for the mid-finger of right hand. This area was the site of a previous digital neurovascular injury, knife wound.

**Frame #2:** Twice daily whirlpool baths with PhisoHex soap and digital exercises are performed. The gangrenous tip of the mid-finger right hand and site of old injury is noted.

**Frame #3:** By the sixth week, is demonstrated a necrotic tip of the mid-finger of right hand with volar fat pad loss to the finger tips throughout.

**Frame #4:** this demonstrates partial finger amputation, mid-digit, right, scarified skin over the dorsum of the right and left hands and contracture of the P.I.P. joints of digits 5,4,3 on the left and 2,3,4,5 on the right.

**Frame #5:** Here are x-ray changes demonstrating periarticular, lytic destructive changes of cartilage and bone at the P.I.P. joints, fingers 5,4 on the right and similar changes on the left hand.





monitor, a near normal pressure may, in eight to 24 hours, change to that of destructive pressure levels, and severe tissue necrosis, especially muscle, occur in a short time.

In 1980, a further paper was published in *Alaska Medicine* on general body cooling. It was stressed that there were many acceptable methods for the emergency care of the hypothermic victim; but regardless of the methods of warming, all demanded the following:

1. Total physiological control of the patient before rewarming.
2. Recognition that the patient is in a "metabolic icebox" with minimal metabolic needs as compared to a state of normothermia, and most probably was hypovolemic and dehydrated, all conditions to be corrected before or during rewarming.
3. It was stressed that electrolyte and acid-base imbalance (most always acidosis) must be corrected. Special concern was to be given to the level of potassium, since probably in deep cooling, and certainly in severe freezing, cell wall permeability is increased. In this event, or in the event of cell destruction, potassium leaks out into the extra-cellular spaces, leaking too, from injured vascular endothelium. If the resulting hyperkalemia is not corrected (especially if enhanced by rapid rewarming), cardioplegia may occur, resulting in sudden death. It has also been helpful to us, besides all of the other monitoring devices in treating severe hypothermia, to begin measurement of cerebral electrical activity by a portable EEG unit to give indication of brain death. (Fig. 24)

Throughout all of the papers in this Journal edition, there remains obvious unsolved problems, namely: the poor results when freezing injury is superimposed on immersion injury or hypothermia; when the epiphyseal plates of children are subject to freezing; and one of the most disastrous events of all, of a freeze-thaw-refreeze

## Accidental Hypothermia

**Problem** : A cold patient in a Metabolic Ice Box: Alive?!

**Purpose** : Bring victim to a physiological responsive state = under

**of Care** : homothermic control — Living.

**Consider** : The higher the temperatures of the thawing methods, the less time you have to direct, control, obtain and maintain the normal physiological state.

**Method** : Rewarm only under total patient problem control.

Fig. 16.

## Accidental Hypothermia

### The Problem

- 1) Lowered Core Temperature
  - 2) Decreasing function of Metabolic System
  - 3) Dehydration
  - 4) Loss of Caloric Reserve
  - 5) Enzyme System Dysfunction
  - 6) Hypoxia of tissues and transfer to anerobic metabolism
  - 7) Metabolic Acidosis
  - 8) Renal Dysfunction
  - 9) Increasing loss of Neuro-regulation
  - 10) Fluid shifts and Electrolyte imbalance
  - 11) Metabolic Ice Box
  - 12) in field — Death from total system cessation
- After rescue — Life or death dependent upon gradual orderly controlled reorganization of organ systems.

Fig. 17.

## Accidental Hypothermia

### What are we attempting to do

- 1) Safely, under control, rewarm the cooled (cooling) body, and elevate core temperature.
- 2) Obtain total physiological control of the patient by:
  - a) Adequate airway control
  - b) Restoration of fluid electrolyte imbalance
  - c) Correction of dehydration
  - d) Correct acidosis — alkalemia
  - e) Restore renal function
  - f) Develop adequate intravenous access (multiple)
  - g) Properly monitor the heart, vital signs, fluid intake and output
  - h) Recognize and/or treat all other conditions prohibiting immediate recovery.

Fig. 18.

## Accidental Hypothermia

### Hospital Care (Warming)

1. Warm by your most familiar, effective method
  - a) Spontaneous Warming — Controlled
  - b) External Rapid Rewarming (90-106°F)
  - c) Peritoneal Dialysis
  - d) ? Both b and c above
  - e) Extra Corporeal (Heated) Circulation (Blood)
    - 1) Heart Lung Apparatus
    - 2) Hemodialysis
    - 3) Other technology
  - f) Anesthetic Breathing Apparatus Support, with Total Control
  - g) Warm Inspired Air with Total Control
  - h) Thoracotomy: Lavage: Controlled?
  - i) However: Have Total Control!!!!

Fig. 19.

injury. This freeze-thaw-refreeze injury is of major concern at altitude, as in rescue from Mt. McKinley, where other than refreeze injury, compression injury may occur above the level of 15,000 feet. At that altitude, the barometric pressure, near one-half an atmosphere,

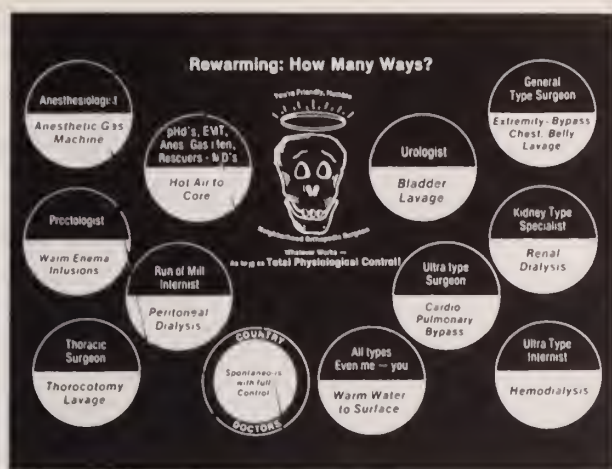


Fig. 20.

permits expansion of cellular stocking or bootliner material, such as neoprene. Non-pliable leather or plastic outer boots, unable to expand, allow the increased pressure from the underlying cellular expansion to be directed downward against the soft tissue of the foot. This compressive force deprives the foot of required microcirculation; ischemia develops, followed by local circulatory failure and freezing.

Still unsolved, too, is the case of the individual with what appears to be straight-forward, superficial or deep freezing injury, who after rapid rewarming in warm water at the stated temperatures, or even lower temperatures, gradually develops tissue demarcation and gangrene, and requires amputation of the part. Suspect here, perhaps, is severe endothelial damage or reperfusion injury.

Treatment can generally be divided into two categories: pre-thaw and post-thaw.

## PRE-THAW TREATMENT

1. The frozen part must be protected to avoid trauma and the risk of irreversible injury at the frozen-nonfrozen interface, which may result if motion occurs at that level, fragmenting partially frozen tissue.
2. The frozen part should then be thawed in a whirlpool bath or a tub at 38-41 degrees C. At Providence Hospital in Anchorage, Alaska, an electrically operated hoist (crane) has been in use since 1963, which lowers the patient's whole body into a Hubbard tub, with whirlpool. This method has also been used for the rewarming of hypothermia victims, and to warm and thaw the combined injury of hypothermia with extreme freezing. The thawing is completed when the distal tip of the thawed part flushes.
3. The thawed part should not be massaged. Rapid rewarming must not be used if the part has been thawed previously.

## POST-THAW TREATMENT

1. When injury is severe and deep, and hospitalization is required, the extremities are kept on sterile or clean sheets, with cradles over the frostbitten part to avoid trauma and pressure. This is not necessary for the upper extremities, which may be placed on sterile or clean sheets, placed over the chest or trunk. Arms and hands are often elevated in stockinette sleeves to permit drainage and encourage digital motion, and decrease dependent edema.
2. Treatment is open, not occlusive, without the use of wet dressings, unguents, ointments or petrolatum gauze.
3. Whirlpool baths are utilized twice daily for 20 minutes at a time, at temperatures of 32-35 degrees C. (90-95 degrees F.).
4. Surgical soaps, such as hexachlorophene, 4 percent chlorhexidine gluconate, and povidone iodine, are employed in the whirlpool.
5. Occasionally, following Moyer's methods for burns, 0.5 percent silver nitrate solution may be lavaged over the area of frostbite. The end result is similar to that produced by the surgical soaps; with Moyer's solution epithelialization is similar, with one outstanding difference: pain is reduced, and infection, even superficial, is much less obvious using the silver nitrate solution.

The whirlpool clears the debris from the injury and removes superficial bacteria. The tissues are debrided without trauma by the whirlpool action at a time when they are physiologically prepared for the separation of the viable tissue from the overlying eschar.

6. Recently, 1 percent silver sulfadiazine solution has been utilized on open wounds secondary to freezing injury, when severe drying and premature blister rupture have occurred, usually followed by superficial infection.
7. Generally, blisters are left intact because the contents are usually sterile, as is the underlying tissue. The blisters are debrided or trimmed only if they are infected and contain purulent material.
8. Escharotomy should be performed on the dorsum or lateral aspect of the digits when the eschar is dry and has firmed sufficiently to have a cast effect on the digits, limiting their joint motion. Digits will be debrided further in the whirlpool, without prematurely exposing underlying epithelializing tissues.
9. Debridement or amputation should be delayed until sufficient time (often 15-45 days) has elapsed to demonstrate a line of demarcation.



**Fig. 21. Case #511-76 #1**

**Frame #1:** A McKinley climber, with 4 to 5 days cold exposure at 5,800 meters, severe dehydration, freeze-thaw-refreeze injury and caloric depletion. Ambient temperature was -35 degrees C., severe winds.

**Frame #2:** Patient after helicopter evacuation from 4,267 meters, left foot is in the climbing boot with zippered neoprene liner, ice throughout the boot.

**Frame #3:** The feet totally frozen to the malleolar level demonstrate the zipper indentation of the neoprene sock. This is a phenomenon not uncommon on high mountain freezing injury. Unlike mountains near the equator, the barometric pressure at 4,572 meters to 4,877 meters approaches one-half atmosphere. As a consequence, cellular material in the boot or sock at this altitude expands, and if the outer boot (leather or plastic) is rigid, the pressure is directed backward to cause compression of the foot and occlusion of the underlying vessels. This contributes to loss of circulation, compartment swelling and pressure increase, and freezing injury.

**Frame #4:** Isotope studies almost two weeks post-injury demonstrate loss of capillary perfusion to the level of heel pads and the mid-tarsi.

**Frame #5:** Infection, minimal because of whirlpool twice daily. Two months has elapsed with dry mummification of tissue and complete demarcation.

**Frame #6:** At three months, the guillotine amputation at the tarsal level, followed by split thickness skin cover has readied the feet for revision amputation and pedicle flap cover as necessary.



**Fig. 22. Case #511-76 #2**

**Frame #1:** Both hands, still frozen, the fifth day of exposure (a freeze-thaw-refreeze injury). An adequate history was limited because of severe disorientation of this patient and his companion.

**Frame #2:** The hand several hours post-thaw demonstrate no distal blebs, early mummification, flattening of the finger tips and digital cyanosis, all poor prognostic signs.

**Frame #3:** 24 hours post-thaw. Gangrene and mummification of the digits is almost complete. This severe, early change is pathognomonic of a freeze-thaw-refreeze injury or thawing with excessive heat, especially the former. Fasciotomy revealed no viable tissues in digits or distal palm.

**Frame #4:** The isotope scan demonstrates total loss of capillary perfusion, just distal to the metacarpophalangeal junctions.

**Frame #5:** Amputation assisting the spontaneous demarcation was performed to the level of viability, guillotine style.

**Frame #6:** The final result, a true disaster but permitting function with prosthetic use. On the right foot, amputation left only a portion of cuboid and cuneiforms. On the left foot, only the calcaneus, talus and navicular remain. On left hand, amputation is mid-metacarpal level for the lateral four digits and at the metacarpophalangeal joint of the thumb. On the right hand, amputation is at the distal metacarpals for four lateral digits and at the metacarpophalangeal joint of the thumb.



mummification and tissue death, with no danger of further retraction of tissues.

10. Overwhelming infection, often found in freeze-thaw-refreeze injuries, or in extremity trauma complicated by freezing, may result in overwhelming sepsis, requiring immediate amputation to avoid toxic shock.
11. If the extremity has remained in a frozen state for some considerable time, even rapid thawing and general supportive care may not be effective in restoring the circulation, and a condition similar to anterior tibial compartment syndrome may be clinically demonstrated. This problem may require fasciotomy. The condition can be determined either clinically or by measuring compartment pressures, by the use of arteriography or the injection of isotopes such as technetium 99m to demonstrate the state of cellular perfusion.
12. Isotope studies have been performed as a diagnostic aid of cellular perfusion for over 30 years. Doppler ultrasound has also been used as a vascular study tool. Interestingly, Thermal Unit patients at Providence Hospital, with Doppler evidence of good pulses in the distal extremities (distal digital vessels), have had conflicting isotope evidence showing failure of extremity perfusion in the same area. In all cases but one, the isotope study was the accurate one. Evidently, large digital vessels may remain patent for a short while, even when the deep capillary system is blocked. See figure 1.
13. Failure of sophisticated tools has also been demonstrated in the use of devices for measuring compartment pressure. If one's clinical judgment and experience advise that an immediate fasciotomy is required, whereas the pressure transducer indicates that the pressure is high but not lethal, or indicates a marginal reading, it is often better to trust one's own judgment. A later measurement may indicate sudden pressure increase. A delay in performing the fasciotomy may be disastrous. This diagnostic problem may be avoided by the use of continuous pressure monitoring. However, there are many pitfalls. The monitoring device is still only a machine, and if one's own studied, concerned opinion is that the fasciotomy should be performed, it may be best to rely on your surgical judgment and release the pressure. (This thought is worthy of repetition in this article.)
14. The grafting of split thickness skin to large granulating areas, or areas where skin cover is considered proper, may be carried out from the third to the 14th day. The results of skin graft are best following thawing by rapid rewarming. The pedicle grafting of full thickness skin is a late

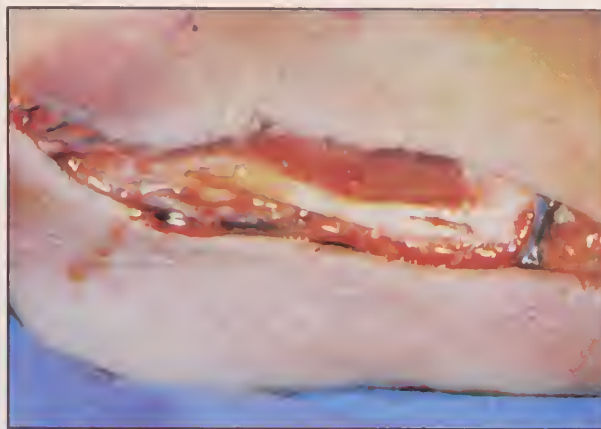


Fig. 23. C-731

Fasciotomy, initial incision, less than six hours post-thawing, demonstrate swollen tissues, with clotting already present in the superficial veins. Operative procedure for increased compartment pressure.

procedure. The use of a mesh skin graft at the time of fasciotomy or soon after, reduces the morbidity and lowers the incidence of scarring and infection.

15. Antibiotics are not necessary, except where infection is deep. Common bacterial organisms found in the injured tissues include Staphylococci, Streptococci, Pseudomonas species, and often an abundance of gram-negative species. Clostridia species are occasionally found. Routine cultures and sensitivity studies are taken, and at the first indication of nonsuperficial infection, not cleared by whirlpool washing, aggressive antibiotic therapy by oral, intramuscular or intravenous methods is utilized. It is again stressed that one of the purposes of the post-thaw whirlpool is continuously to dilute bacterial accumulation on ischemic or necrotic tissues.
16. Cotton pledgets between digits will prevent maceration of tissues. In the presence of severe digital edema, however, pledgets or cotton swabs may compress digital vessels, further compromising the circulation. Bedside digital exercises of all the joints are recommended, and should be done throughout the entire waking day, and Buerger's exercises for the lower extremities are recommended four times daily. Narcotics are utilized sparingly in uncomplicated cases after initial



Fig. 24.

thawing; tranquilizers or aspirin will suffice for pain. In the past, sympathetic blockade, sympathectomy, anticoagulants, vasodilators, alcohol and enzymes have not proved particularly effective.



17. Over the years, many surgical procedures have been proposed for post-thaw care of the frostbite-injured extremity. The benefits of surgery should always be weighed against the possible injury to the regional vascular structures in the injured area. If possible, the surgical approach should improve the prognosis by relieving compartment pressure, increasing joint mobility, limiting infection, or increasing vascularity.
18. In the past, on one occasion, the use of a single man hyperbaric oxygen chamber, delivering two atmospheres of pressure, had appeared beneficial in post-thaw frostbite. Again, however, hyperbarism is not likely to be helpful if the oxygen transport system is blocked by vessel thrombosis, or destruction of vascular endothelium
19. In patients with apparently equal bilateral injury, results of sympathectomy within the first 24-48 hours have demonstrated that while there is no further preservation of tissues, there is a decrease in pain, a marked decrease in edema, much less infection (superficial and deep), and early and more proximal tissue demarcation.
20. Along those same lines of thought, the use of phenoxybenzamine hydrochloride (Dibenzyl-ene®) has been particularly effective given in doses of 10 mg per day, and increased to 20-60 mg per day, depending upon effect and need. The drug is used for vasospasm, and appears to be an effective alpha adrenergic blocking agent. It is, in our program, seen to be an effective medical sympathectomy. It appears to be a trial method of determining whether surgical sympathectomy would be effective. It is important to avoid decreased volume effect from vasodilatation, so the patient should be kept well hydrated while using Dibenzylene.

It should be noted that on occasion, vascular arterial stripping of small vessels in hands and feet is an effective form of sympathectomy, helping not only in perfusion, but in decreasing pain. However, more recently, and still under evaluation, sympathectomy and vasodilators and sympathetic blockade have been determined to be of good effect following fasciotomy. The author suspects that the previous irregular results of these treatment methods often reflected an effort to perform effective sympathectomy, when the problem may have included regional vascular compartment pressure block or distal vascular thrombosis.

21. Pain varies with each individual. Request for pain medication often increases when it is apparent that necrosis of tissue is imminent or present -- having both a physical and psychological cause for

## **FREEZING INJURY : SURGICAL PROCEDURES**

1. **ESCHAROTOMY---ESCHARECTOMY**
2. **BLEB , BULLAE , WOUND DEBRIDEMENT**
3. **FASCIOTOMY**
4. **ARTERIOTOMY**
5. **VASCULAR WOUND REPAIR**
6. **DERMAL GRAFT PROCEDURES**
  - a. **REVERDEN (DAVIS) PINCH GRAFTS**
  - b. **SPLIT THICKNESS SKIN GRAFT**
  - c. **SPLIT THICKNESS SKIN GRAFT -( MESH )**
  - d. **FREE FULL THICKNESS SKIN GRAFT**
  - e. **CUTANEOUS PEDICLE FLAP GRAFT**
  - f. **MUSCLE,MUSCULO-CUTANEOUS VASCULAR FLAP TRANSFER**
  - g. **VERY EARLY DIGITAL DEBRIDEMENT WITH VASCULAR CUTANEOUS FLAPS**

Fig. 25a and 25b.

## **SURGICAL PROCEDURES-( CONT. )**

7. **CONTROLLED SUB-CUTANEOUS BALLOON TISSUE EXPANSION**
8. **GUILLOTINE ( ? MODIFIED ) AMPUTATION**
9. **CLOSED AMPUTATION ( ? CLOSED SUCTION IRRIGATION )**
10. **CLOSED OR OPEN REDUCTION OF FRACTURES,DISLOCATIONS**
11. **JOINT CONTRACTURE RELEASES JOINT EXCISION AND REPLACEMENT JOINT FUSION**
12. **SOFT TISSUE,WEB SPACE RELEASES**
13. **SURGICAL REGIONAL SYMPATHECTOMY**
14. **PERI-ARTERIAL SYMPATHECTOMY MICRO-DIGITAL SYMPATHECTOMY**
15. **EXCISION SINUS TRACT SQUAMOUS CELL CARCINOMA**
16. **TISSUE COMPARTMENT RELEASES CARPAL , TARSAL TUNNEL SYNDROME**

**Fig. 26.** Case 182.

**Frame #1:** Buried under cook shack debris for 79 hours, pinned under snow, one hand buried, able to breath.

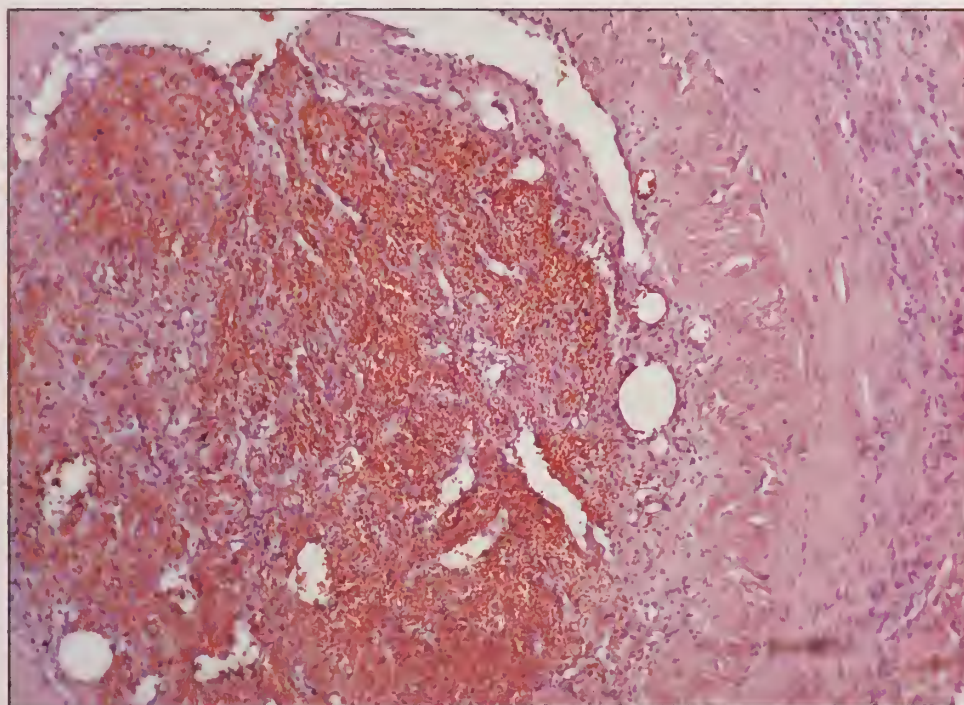
**Frame #2:** Examination in Alaska hospital indicated mild hypothermia, freezing injury of right hand and feet.

**Frame #3:** Patient hypovolemic and dehydrated, mild hyperkalemia, with ph of blood 7.7, possibly from curling ulcer treatment with NG tube extracting H<sup>+</sup> ion.

**Frame #4:** Hand, after thawing, on fifth day, with cyanotic finger tips.

**Frame #5:** Single man hyperbaric chamber flown into Ketchikan by Canadian government.

**Frame #6:** Pre and post hyperbaric chamber, at two atmospheres. Is there a difference? Some immediate flushing of the feet was noticed at the completion of hyperbaric use. (Seen in consultation Drs. Wilson, Ketchikan, 1965).



**Fig. 27.** C-1079-88.

Long standing wet-cold exposure, followed by freezing. Section of small peripheral artery of foot.

1. Adventitia demonstrating non specific inflammation.
2. Tunica media with less obvious inflammation, with spindle shaped muscle fibers.
3. Thickened intima and circumferential muscle fibers.
4. Endothelial disruption growing into a luminal clot, with endothelialization of thrombus.
5. Intra luminal clot being organized with in-growth of fibroblasts and capillaries.



**Fig. 28. Case #ILL. 1**

**Frame #1:** This represents the hand of a patient with Raynaud's phenomenon or one of many thousand individuals who demonstrate a labile vasomotor response to cooling, even if that cooling is by a slight drop of ambient temperature from 68 to 65 degrees F., for example. This condition, more common in women than men, responds to biofeedback training or physiological self-regulation. It is considered a method of preventing as well as treating frostbite or cold injury.

**Frame #2:** Relaxation techniques and biofeedback training utilizing self-regulation permit cool or cold hands and feet to be warmed by reversing the phenomenon of cold-induced vasoconstriction (CIVC).

**Frame #3:** Control of each hand is identified here: vasodilatation of the right hand with thermistor control and evidence of a cold, cyanotic, unregulated left hand.

**Frame #4:** A frostbite patient with necrosis of the large toe is using biofeedback to increase vascularity in the border area of viability.

**Frame #5:** A borderline freezing injury is treated by biofeedback techniques to avoid tissue loss and increase circulation.

**Frame #6:** The technique is particularly effective for hands. They appear to be more easily warmed in most patients than feet. The technique is helpful, too, in warming digits to avoid injury. In groups outdoors, it is used as a means to avoid cold injury and to warm feet where there have been warning signs of loss of local heat.

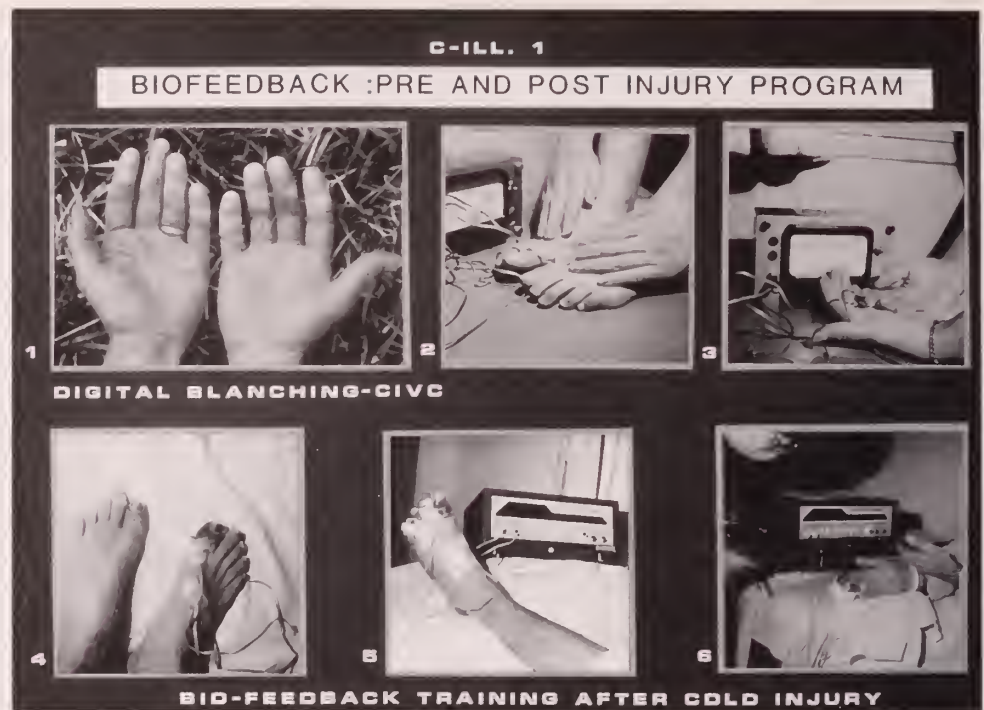
The technique is most helpful in the hospital, too, because it encourages patients who are concerned over the appearance of cyanosis, and early tissue loss. It gives them a means of cooperating and contributing in their own treatment, and has often demonstrated that it can raise skin temperatures from the low 70s to the high 80 degrees F. or higher.

pain and distress. The discomfort, too, varies with the degree of edema, the type of injury and the presence or absence of infection. It is lessened by a healthy doctor, nurse, patient relationship; by adequate physio and occupational therapy; by whirlpool baths; and analgesia as required.

22. In severe cases of freezing injury, and particularly so with the acute stage of post-warming immersion injury, pain relief may be provided with continuous epidural block for 24-48 hours, repeated if necessary. This treatment is especially effective if accompanied by fascial release in severe cases of freezing or immersion injury when increased compartment pressures are present. Helpful to these patients is an anesthesiologist or pain center physician. Choices of epidural medication usually are 0.25 percent Marcaine or 1.5 percent Zylocaine, occasionally supplemented with a steroid such as Depomedrol.
23. In the past decade, biofeedback training has been utilized for the patient with post-thaw freezing injury. It has been helpful, too, as an antistress tool, in the patient presenting with a labile vasomotor response to cold, as a pre-injury

avoidance tool. The biofeedback program (see Kappes, this issue) is also an adequate form of occupational therapy, at the bedside, or as an outpatient since its use is often demanding and repetitive. By its very method, time and effort are required for biofeedback benefit. As the days, and especially nights, are long for the patient hanging on the edge of revascularization versus ischemia and gangrene, the biofeedback exercises fill a time void.

**Note:** Perhaps the most important adjunct in severe hypothermia, freezing injury, immersion injury, or any combination thereof, is to have available a team consisting first of a learned emergency room physician, or any first responder physician interested in cold problems. Included in that group should be a physician from the field of internal medicine; a general, thoracic or cardiovascular surgeon; an orthopedic surgeon; a plastic surgeon; an anesthesiologist; and in the wings, a neurosurgeon or psychiatrist. Availability of a section of Radiology and laboratory facilities is helpful. And, of course, exceedingly helpful are knowledgeable nurses in the Emergency Room area, as well as the thermal care





**Fig. 29.**

If gangrenous change, mummification, or infection is present after frostbite, then any amputation performed (here trans metatarsal) should be closed over suction irrigation, for dilution of bacteria in the area. This method is helpful for metatarsal amputation for the gangrenous diabetic foot.



**Fig. 30. C-222**

An Alaska 'sourdough' came to the Yukon in 1901, lived in most of the gold camps; occupation cook. Age 80. Had frostbite on "lots of occasions" — treated each episode with cold, or warm coal oil, a common Alaska home remedy. Evidence of multiple injuries, bulbous tips, fat pad loss and osseous atrophy.

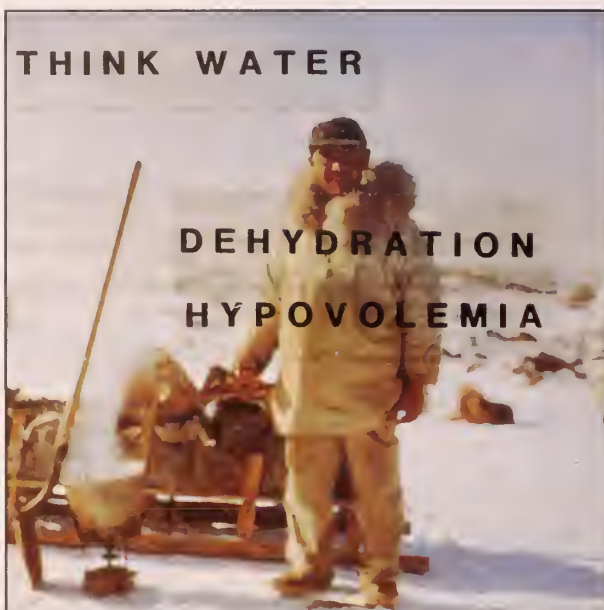
unit or surgical or medical wards to which the patient is assigned.

Suppose wherever you are in Alaska, you have little of the above. Remember, most of your tools are in your head and hands. Any place with a roof, water, heat and a bed, that has a nurse and physician can be a hospital. In Vietnam, a group of Navy and Marine personnel at Dong Ha did stellar emergency care in an old French Cavalry stable! For sure, if you are an Alaskan physician, you should know at least the basics of care for a real Alaskan problem -- cold injury of all kinds -- and have knowledge of prevention, too, as most Alaskan medical personnel, are wilderness travelers and outdoors folks. The limb you save may be your own!

## ADDITIONAL CARE AND AFTER THOUGHTS

1. The diet should be high in protein and in calories, with vitamin supplements as needed.
2. When considered necessary, antitetanus therapy is recommended, particularly toxoid booster for those previously immunized.
3. If amputation must be performed, a modified guillotine procedure at the lower level is recommended, with secondary closure to be carried out at a later date. An interim mesh skin graft should be utilized to close the wound and eliminate drainage and infection, before secondary closure.
4. Superficial or deep infection is often found in the extremity requiring guillotine amputation. Secondary closure after the amputation may be more successful when accompanied by closed-suction-irrigation, with irrigation fluid (0.9% sodium chloride) flowing at 100 ml per hour, with a flush of 50 ml of the preferred antibiotic solution every hour.
5. Dislocations and fractures pose interesting problems, and dislocation particularly should be reduced immediately after thawing.
6. Traction, manipulation or open procedures are used rarely, and only with great care. Fracture treatment should be conservative until the post-thaw edema is eliminated. It may be that a well-padded plastic mold is the best method of treatment until edema ceases. If open reduction of fractures or dislocations is required, great care must be taken to avoid further vascular injury. Post-operatively, the operated part, in a plastic posterior mold, may still undergo whirlpool therapy and active digital exercises. The prognosis of this combined injury is poor, because added to the injury to the regional vascular supply from fracture trauma, is the superimposed freezing injury.





**Fig. 31.**

A first consideration in evaluation of hypothermia, freezing or immersion injury in Alaska, is the determination of the state of hydration, as well as, electrolyte and acid-base status.

7. It is here also that fasciotomy may be required to relieve the deep structure pressures.
8. Fluids are encouraged, dehydration is to be avoided, and electrolyte balance should be maintained. Smoking is discouraged, but alcohol may be permitted.
9. The cartilaginous structures in children, particularly the epiphyseal plates and nonossified carpal and tarsal bodies, are susceptible to cold insult and injury. Total necrosis of these organs is rapid, and at present, regardless of the methods of thawing and post-thaw care, the injury is apparently irreversible.

Adults, too, may demonstrate significant changes in bone and cartilage after freezing injury. This change is not apparent at the time of original insult. Therefore, for quite a few years, an x-ray (flat plate) of hands and/or feet has been taken soon after the freezing injury onset. Those films, if repeated in five to six months, are most often unchanged from the initial x-rays. However, on x-rays from six to 18 months, often interesting radiographic changes appear. They range from large areas of avascular necrosis of bone to small punctate, lytic changes, often peri-articular in position, especially in involved digits. Having obtained early x-rays for comparison, allows one to rule out chronic disease as gout, rheumatoid disease, or other benign or malignant conditions presenting in this fashion. Should the patient later sustain trauma to those parts, with the inadvertent discovery of such lesions, it is helpful to have such an x-ray series on file, identifying the pre-existing x-ray change due to freezing.

All of the above is a basic program, to which the physician may add any other therapy of choice. Despite the best intentioned treatment (regardless of thawing method, indicated drugs or surgical care), some results are unexplained disasters. These poor results may be due to extended depth and duration of freezing, repetitive freeze-thaw-refreeze injury, underlying circulatory deficit or to some other cause. The post-injury state may demonstrate freezing injury with post-thaw vasoconstriction, arteriovenous capillary thrombosis, and severe cellular destruction.

Being aware of all the choices will help in the selection of the appropriate drug therapy. Anticoagulants (heparin), vasodilators (tolazoline hydrochloride) or hypotensive adrenergic blocking agents (guanethidine, reserpine), including sympatholytic drugs (phenoxybenzamine hydrochloride) may aid in the initial phase of care, especially in the absence of deep thrombosis. Plasma volume expander such as low molecular weight dextran used early is thought to prevent, diminish or reverse red cell aggregation in the capillary tree. The use of thrombolytic enzymes is being studied; their use, though, is limited at this time, especially because of the hemorrhagic effect on an already damaged microcirculation from freezing. The risk of hemorrhage and lysis of fresh fibrin obviously limits their use where there is associated trauma, especially injury to head or spine, in which a cerebral or spinal cord bleed may be disastrous. The use of these drugs and those summarized below, may require special local and regional techniques. There is currently little help for the problem of severe or total cellular destruction. And as for the case of the hyperbaric oxygen transport, no drug will be regionally effective, not even antibiotics if the vascular tree is no longer patent. One wonders whether DMSO should be approved by the Food and Drug



**Fig. 32. C-1083-88**

For an early prognosis of the cold injury, thermography, combined with isotope studies is helpful. Evaluation of superficial (thermography) and deep (Technetium-99m) tissues are permitted. Both are an aid in determining the effectiveness of therapy, as time continues.



**Fig. 33.** C-134 12-13-63

Three week post injury, after immersion in a creek to hips. (ambient temperature -5 degrees F (-20 degrees C.). Exposure in walking back to car, 3 hours. Partial warming in vehicle followed by rewarming in warm water in hospital. Feet frozen bilaterally. Epidermal eschar present, with viable tissues below. Full thickness loss on right dorsum.



**Fig. 34.** C-134.

Five weeks post injury, after successful skin graft. Intrinsic muscle loss and contracture, with partial loss of large toe tip. Volar foot pad loss of toe tips.



**Fig. 35.** C-134-63

X-ray of right foot seven weeks post injury reveals no definite degenerative changes in phalanges.



**Fig. 36.** C-134-63.

After two 1/2 years post freezing degenerative changes have occurred in the subarticular regions of MP and IP joints. There is presumed cartilage destruction with loss of joint spaces. All accompanied by limited range of joint motion.

Administration of efficacious drugs might be attached to its structure, since it appears to be an effective penetrant.

Anti-inflammatory drugs, aspirin or ibuprofen for example, have been given to prevent or decrease thrombosis following progressive dermal ischemia after arachadonic acid cascade, that permits the formation of prostaglandins, thromboxane and vascular clotting.

## SUMMARY

The drugs used in frostbite injury care are: Plasma volume expanders (low molecular weight dextran); vasodilating agents (tolazoline hydrochloride); hypotensive agents (guanethidine monosulfate, reserpine); hemorrhheologic agents (oxpentifylline); calcium blocking agents (nifedipine); sympatholytic agents (phenoxybenzamine hydrochloride); anticoagulating agents (heparin); thrombolytic enzymes (streptokinase, tissue plasminogen activator -- TPA); an industrial solvent





Fig. 37. C-134-63.

Six 1/2 years after freezing, MP joint degenerative changes have increased, with increase in sclerotic avascular bone in area of first MP joint, and increased loss of IP, MP joint motion.



Fig. 38. C-134-63.

After 14 1/2 years, the narrowing of all joints persists, with periosteal joint areas of MP and IP joints demonstrating fibrin filled destructive lesions. Clinically the PIP joints of toes 3,4,5 are immobile. Hammer toe deformity has developed at DIP joint second toe and on toes 2-5 on left foot. MP joint motion, however, totaled 25 degrees on the right.

(dimethyl sulfoxide -- DMSO); anti-inflammatory agents such as nonsteroidal drugs, and acetylsalicylic acid, Ibuprofen.

As yet, no clear treatment policy has been determined for preventing injury secondary to the formation of oxygen free radicals, damaging neutrophils or reperfusion injury. The role of oxygen free radical scavengers and factors causing reperfusion injury is unclear at this date.

Since that first reported series of 51 patients in 1960-61, 1,282 patients have been seen. Of that number, 1,026 had a diagnosis of frostbite; 151 were diagnosed as hypothermia; and 105 diagnosed as immersion injury.

As Table 1 demonstrates, 54 patients in a hypothermic state had frozen extremities, while another group of 15 hypothermia victims had associated immersion (wet-cold) injury. Twenty-five immersion injury victims had superimposed freezing injury. Six hypothermic patients had pre-existing immersion injury with superimposed freezing injury.

Table 1.

	Frostbite	Hypothermia	Immersion Injury
Frostbite	1026	54	25
Hypothermia	54	151	15
Immersion Injury	25	15	105

In another paper in this edition, the types of freezing injury found in Alaska are listed, as well as the most common thawing methods used.

From a review of these many cases, what results may be anticipated following treatment of freezing injury?

The prognosis in freezing injury seems to be determined as follows:

- I. The prognosis is best when
  - a. The duration of freezing is short;
  - b. The depth of freezing is superficial;
  - c. Freezing is not associated with fracture, hypothermia, immersion injury or other trauma;
  - d. Thawing is by rapid rewarming;
  - e. Blebs are present, are clear, are large and extend to the distal tips;
  - f. There is an early and rapid return of capillary perfusion, especially as demonstrated by technetium 99<sup>m</sup> radioisotope studies.
- II. The prognosis is uncertain when
  - a. Thawing is spontaneous;
  - b. The frozen state is of long duration;
  - c. Freezing is superimposed on fracture or dislocation, or is associated with severe soft tissue trauma;
  - d. Freezing is superimposed on conditions of preexisting disease with vascular deficiency (example, diabetes), or hypothermia with associated hypovolemia and dehydration, or hypoxia.
- III. The prognosis is poor when
  - a. Thawing is delayed (ice, ice water, snow, friction massage);
  - b. Thawing is by excessive heat (greater than 49 degrees C. (120 degrees F.));

- c. Post thaw blebs are proximal, dark, hemorrhagic, and do not extend to the distal phalanges;
- d. Necrosis is early, with early advent (4-5 days) of mummification or liquefaction necrosis of the involved part;
- e. There is obvious loss of cellular perfusion as identified clinically and confirmed by technetium 99<sup>m</sup> studies on early and repeated views;
- f. There is a freeze-thaw-refreeze injury; and
- g. There is suspicion of reperfusion injury.

Tissue freezing is almost always followed by some residual loss of anatomy or function; slight though that loss may be. Post-freezing sequelae may be categorized as 1) transient or 2) long standing.

There are generally, in severe freezing, sequelae. Post-freezing sequelae are:

- I. Transient -- early signs are
  - a. Hyperhydrosis;
  - b. Hypesthesia, or anesthesia of digits;
  - c. Limitation of motion IP and MP joints;
  - d. Joint swelling, IP and MP joints;
  - e. Edema;
  - f. Thin, fragile epidermis in involved area;
  - g. Nail loss;
  - h. Intrinsic muscle atrophy;
  - i. Fat pad atrophy of the distal tips.
- II. Long standing sequelae, usually permanent are
  - a. Deep fixed scars over the affected areas;
  - b. Atrophy or fibrosis of intrinsic musculature;
  - c. Contracture of digital joints such as hammer and claw toe;
  - d. Volar fat pad loss;
  - e. Hyperesthesia, distal tips with increased sensitivity to cold and heat;
  - f. Decreased proprioceptive sense of the digital tips;
  - g. Permanent nail deformity;
  - h. X-ray evidence of peri- and subarticular lytic destruction of bone and cartilage, of phalanges especially;
  - i. Avascular necrosis of bone, especially phalanges, metatarsi and tarsi;
  - j. In children, epiphyseal necrosis or total destruction of physis, epiphysis, with joint and phalangeal deformity, angulation and shortening;
  - k. Chronic ulceration, infection and osteomyelitis;
  - l. Decreased capillary perfusion;
  - m. Rare finding of squamous cell carcinoma in a persistent sinus tract;

- n. IP joint immobility or fusion;
- o. The ultimate of long standing sequelae, amputation.

At the very beginning of our program, patients were hospitalized, for moderate or severe injury, and remained in the hospital for many days or weeks as revascularization or necrosis and demarcation occurred. The exceedingly high cost of such hospital stays, and the fact that so many of our patients have little or no medical insurance, or are on Medicaid or Medicare, caused us to re-evaluate cold injury care. (This is not a concession to the utilization review committees.) As soon as the patient is able, he is transferred home, or to a nearby out-patient care facility. We convert the bedroom and bathroom into a hospital ward by renting or purchasing a Whirlpool pump for the bathtub, arranging for all drug use at home and following the patient as an outpatient until discharge. Few ill effects have followed this method when the patient and family or friends are cooperative. The program is even more effective if the patient is supervised by visiting Home Health Care personnel.

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# COLD AND FREEZING: A HISTORICAL CHRONOLOGY OF LABORATORY INVESTIGATION AND CLINICAL EXPERIENCE

by William J. Mills, Jr., M.D.<sup>(1)</sup>, James O'Malley, M.D.<sup>(2)</sup>, Bruno Kappes, Ph.D.<sup>(3)</sup>

Over a year ago, I<sup>(1)</sup> was invited to help prepare a segment of a chapter in the revised edition of the *Handbook of Physiology*. The chapter was to be entitled "The Limits of Tolerance to Hypothermia."

In the process of preparing for this study of the effects of freezing, I had a grand opportunity to obtain the original, reprints, or copies of the original articles published by the many outstanding authors writing on cold matters. But even better still, I was given the opportunity to read each article thoroughly. I thus demonstrated the wisdom of collecting papers and reprints that were often filed away unread, the benefits of access to the University of Alaska and University of Washington Health Science Libraries, the pleasure of wandering through my own medical library, and the extra grand leisure of semiretirement, allowing such activity.

In this paper, cold and freezing, I have condensed pertinent information and findings of those papers, mostly investigative studies, that I felt might be helpful to anyone interested in the further study, for whatever reason, of cold injury.

Like most papers, no matter the length, the very best segment for the reader, is the bibliography. The bibliography is the key to your own interpretation and evaluation of all the information presented. And possessing that key is your own opportunity to unlock the closed doors that still inhibit more desirable results in the care of freezing injury, or other cold problems.

## INTRODUCTION

Injury due to freezing has been an unwelcome problem over the ages, usually associated with mountain warfare, winter military campaigns, or the civilian catastrophe of deprivation of shelter, food stocks and warm clothing following in the wake of military conquest. Over the centuries, natural calamities (famine, earth-

quake, pestilence and fire) left many communities, world wide, exposed to harsh winters.

Many of the experiences that have decimated armies and civilian populations in the past have been documented in considerable detail, findings made available for study by modern military leaders and their medical departments, by cryobiologists, physiologists and clinicians concerned with cold injury (3,29,35,54,56,88, 89,90).

In more recent times the reports of freezing injury in the nonmilitary population has increased, involving even the homeless on our streets, and has progressed to include skiers, skaters, hunters, snowmachiners, mountain hikers and climbers, as the popularity of winter sports has increased (31,51,52,54,55). And now, as the explorations of man explode into the wilderness throughout the world, searching for oil, minerals, forest, and the bounty of the oceans, it is more appreciated that hypothermia, immersion injury, and frostbite are known injury risks of those working, as well as living, in the Arctic and sub-Arctic areas. This is particularly true for those who work the circumpolar seas of the Arctic and Antarctic oceans, and the cold northern seas of the Atlantic and Pacific oceans.

Paton (64), in an excellent summary of the pathophysiology of frostbite, noted that "frostbite is as old as history itself." And indeed, as an extensive bibliography demonstrates, particularly during periods of war, cold injury has played a paramount role in the outcome of military operations throughout history. A review of the world's literature on cold injury would indicate the casualties to be literally in the millions, (3,8,29,35,46, 54,57,63,89,90) resulting in destruction of tissue, loss of function, neurocirculatory loss, amputation minor and major, and death.

Over the ages, the travail and disaster befalling armies battling cold, as well as enemy forces, has been documented for posterity; those lessons and that knowledge learned by a few, but sadly disregarded by many. Unfortunate though it be, for millions of cold victims, from the time of Xenophon (89) (400 B.C.), to the cold injured on the Golan Heights in the Israeli winter war of 1973 A.D. and the following British-Argentina involvement in the Falkland war of 1982 A.D., little success was

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noted in preventing serious cold injury occurrence. Freezing and other cold-related injuries have remained wicked and unrelentingly disastrous military and civilian hazards, for at least 2000 years of recorded history. Despite this record, wartime experience with cold has advanced our knowledge of cold injury, in at least two directions. First, from the varied cold injuries, especially freezing injury, incurred by massive numbers of troops, much clinical experience resulted, allowing for new, innovative and comparative treatment regimens. Though unethical it would be to experiment on humans, with perhaps one method for one extremity and another for the other, the sheer force of numbers of injuries with their emergency self-treatment and directed-care methods, permits much clinical evidence to accumulate. And as much is learned from studying bad results as good, when cases are numbered in the thousands.

Secondly, military medical personnel in the field, and clinical and laboratory investigators in research laboratories and hospitals were given impetus and encouragement, as well as funding from a multiplicity of government and private sources to provide insight into the etiology, pathophysiology, and treatment of cold problems.

It is fitting to review some of the past accounts by military surgeons of cold injury, for from the tragedies of beaten armies has come direction for future research. Those armies, overwhelmed by severe cold, harassed by an unrelenting enemy in pursuit, often were without water, food, clothing and shelter and had usually in retreat abandoned most of their equipment. Unable to retreat in proper order, unable to properly care for the wounded, or warm the cold victims, those beaten troops have left us with data regarding the cause and effect of cold, and vivid evidence of effectiveness or failure of field emergency care and treatment — and left to those interested, even today, ideas for further investigation.

Prior to WW II and the Korean war, little information regarding the basic physiological response to cold was available to the interested American physiologist or clinician. One reason was the unfortunate fact that in America prior to WW II, little clinical or scientific data from Europe, Russia, or Japan was available in translated form. Further, clinicians and physiologists as a rule did not read each other's journals as extensively as today. Consequently, the massive clinical and investigative material available prior to WW II from Europe and Asia, was little known.

As an example, until the translated WW II works of Killian (German) (29), Arieu (Russian) (3) and Yoshimura (Japanese) (90) were published, it was not generally realized that the medical departments of their military services had utilized rapid rewarming for frozen extremities.

The period following WW II and the Korean war, found an exciting and rewarding liaison between the clinician in the field and in the hospital on the one hand, and the laboratory based physiologist, biochemist, biophysicist and cryobiologist on the other. The two previously aloof groups now consulted each other and exchanged views, permitting rapid understanding of the pathology and pathophysiology of the victims exposed to cold and the injury(s).

This interchange furnished the clinician with the results of drug, surgical and manipulative procedures first performed on laboratory animals, and permitted the physiologist to perform previously untested surgical or medical procedures that appeared to have promise, also utilizing laboratory animals (18,23,47,51).

## GENERAL KNOWLEDGE

Highly recommended to the student of cold injury are the following texts or monographs which form a nucleus of military and civilian research activities. Each explores, reports, and summarizes a concept of pathophysiology, treatment, and a pattern of injury sequelae. The monographs and texts are summarized in chronological order, as the contents impacted the scientific and medical communities.

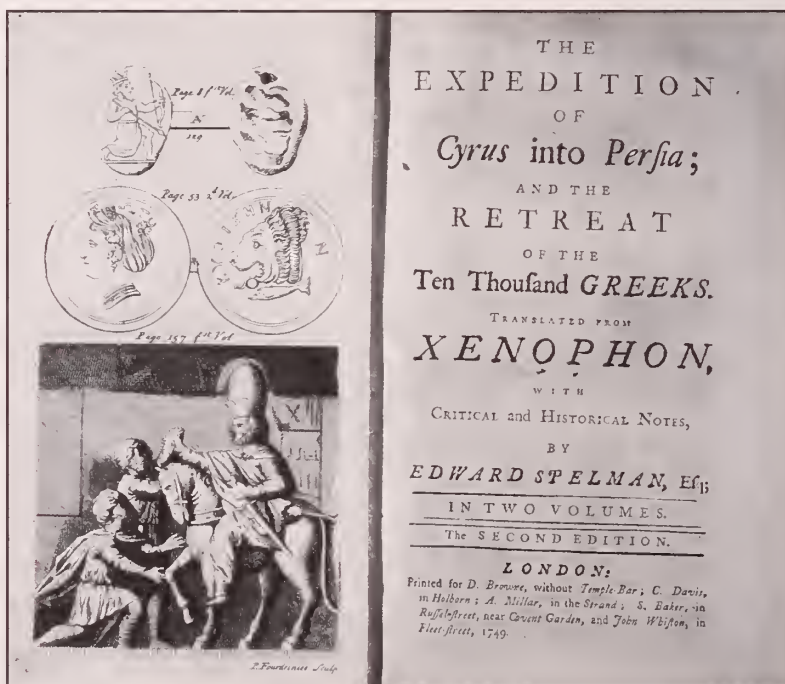


Fig. 1.

One of history's oldest accounts (Fig. 1.) of an army decimated by cold was that of Xenophon, who in 400 B.C. led ten thousand Greek soldiers from Sardis to Babylon and back, through the mountains of Armenia, battling the hazards a retreating and disorganized army faces when pursued by the combined unrelenting foes of severe cold weather and harassing enemy forces. Warmth was obtained by campfire heat, friction massage of the body and body parts with greases, oils, and unguents, and preservation of heat by covering up in snow. Amputation and death from exposure was common (89).

An even more detailed, factual, and vivid description of the effects of cold on a retreating army was related by Larrey (35), the chief surgeon of the Grand Army of Napoleon in the retreat from Moscow, 1812-1813. His memoirs gave precise description of freezing injury, and its etiology, including data on general body cooling. (Fig. 2.) He had recommended slow rewarming, or delayed warming with ice and snow techniques, and friction massage, all out of favor now, although accepted for well over one hundred years after his reports. The monograph is replete with the problems of prevention and care involving massive numbers of troops -- in an army where over eighty percent of its force perished from cold and cold-related problems. Much can still be gained by a study of this classic. Larrey disapproved of rapid rewarming, and his words discouraged the use of rapid thawing for over one hundred and fifty years. He did note the disastrous effect of excessive heat (as documented in recent reports (51,58) apparently recognizing that frozen extremities, warmed in the close proximity of bivouac fires, sustained a second thermal injury, a burn, with disastrous effect. For the student of freezing injury, this monograph is highly recommended.

Monographs of the experience of the military surgeons of modern armies soon appeared following WW II. Wayne and Debakey (88) editing a comprehensive review of the United States Army experience in that war, 1) pointed out that the lessons of previous wars were poorly understood and often forgotten by WW II military surgeons; 2) reported and discussed the seventy-one thousand cold casualties in the European theater; 3) recorded the new syndrome of high-altitude freezing in air crewman (freezing and hypoxia); and, 4) stated that cold injuries were due to the intensity of combat, wet cold, inadequate clothing and lack of troop education. Injuries were to be considered a calculated risk. It was the stated hope of the authors "that if their volume was read well, there would be no need for problems in future wars." But as was later found in Korea (1951-52) in the Yom Kippur war in Israel (1973) on the Golan Heights of Syria, and in the Falkland war (1982) weather, enemy action, and military demands and unforeseen events determine the effects of wet and cold.

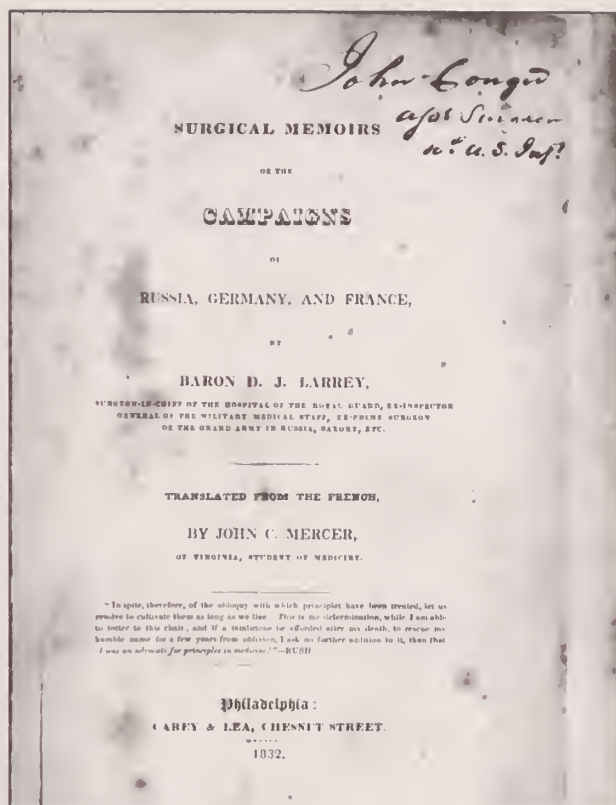


Fig. 2.

A comprehensive monograph by Killian (29) related the cold problems of the axis forces in WW II, with a discussion, still of concern today, of slow as compared to rapid rewarming, the latter being preferred by many German military physicians, despite slow thawing methods being the European dictum.

Killian reminded his readers of Vant Hoff's rule "that under conditions of hypothermia, local or general, that metabolic processes are slowed down so that oxygen demand of tissue is reduced, thus prolonging survival of ischemic tissue." In much later reports, Mills described this as "being in a metabolic icebox" (54,57). This condition he characterized as the victim being in a mid lethal state, so that further exposure would result in death as cooling of vital organs continued without intervention and warming. Warming, however, in this state, if not controlled physiologically, would often result in death because of uncorrected acid base imbalance, electrolyte imbalance, usually acidosis, with hypovolemia and dehydration. Warming of the hypothermic victim, when frostbite also is present, may release potassium from increased cell permeability or cell destruction. The often sudden high level hyperkalemia may result in cardioplegia and death.

From Russia, Ariev (3) in a little known, but true classic on cold, reviewed the current European pathophysiology concept, listing the development of cold injury descriptive nomenclature and classification of





Fig. 3.

Harassed by the enemy, overwhelmed by cold, the French army retreated from Moscow in 1812-18, many attempted to swim the Berezina River, falling victim to hypothermia, as well as extremity freezing. Thousands of French soldiers perished due to starvation, dehydration, hypothermia and freezing — and unrelenting Cossack attacks on their flanks. The army of Napoleon lost over 80% of its troops, one of winters' greatest triumphs over a military force. Copyright photo "Musée De L'Armée, Paris."

frostbite, as well as recommending that rapid rewarming be the method of choice in the thawing of freezing injury.

This report was followed by a report from Japan, when Yoshimura (90) proposed thawing methods similar to Killian and Arieu.

A further report of low temperature investigation, encouraged because of the needs of military surgeons, was the monograph, *Man in a Cold Environment* (11), by Burton and Edholm of the Physiological Society. This monograph originally proposed to review experiences in cold in WW II, but was later changed to include all aspects of cold, listing an investigative bibliography to 1955, and is included here as a "must reference" in the field of cold study -- as a source development of the fundamental basis of the scope of the "cold problem." (Sponsored by the Defense Research Board of Canada.)

Viereck (86) edited the proceedings of an Air Force sponsored symposium on frostbite in 1964, which brought together clinicians, laboratory investigators and pathologists and others experienced in cold.

An outstanding textbook soon followed, edited by

Meryman (47), that gathered together the works of eighteen prominent physiologists, biophysicists and cryobiologists delving into the theoretical basis of freezing injury. It is a definitive review, and a framework text on the comprehensive background of biological freezing which includes studies of the physical and chemical basis of injury in single-cell micro-organisms.

In 1975, LeBlanc (36) published another landmark monograph, *Man in the Cold*, unique in that his observations were made primarily on humans, rather than from laboratory animals. In that monograph, LeBlanc in the section on frostbite graphically describes the cooling, freezing and post-freeze states, and reviews data regarding the pathophysiological stages of vascular disturbance, membrane permeability, and post-thaw edema formation.

## INVESTIGATIVE PUBLICATIONS

As this is a review article on freezing, it is perhaps pertinent now to repeat Meryman's concept of freezing



Fig. 4. Reprinted from Cryobiology, 1966, Academic Press, pp. 325, from The Freezing of Animal Tissue, Love, R. Malcolm from Meryman, 1963. Copyright permission - Federation Proceedings 22, 1963. Federation of American Societies for Experimental Biology.

1. Some 'substance' in fluid of the extracellular space permits a nucleus of ice to form.
2. The extracellular solution has been concentrated from freezing, and the resultant high osmotic pressure in the extracellular allows intracellular water to diffuse through the cell wall.
3. The continuous growth of extracellular ice permits mechanical pressure to be exerted upon cells, compressing them. This results in progressive cell dehydration, a condition more damaging to the cell wall than the mechanical effect of the ice crystals.

(45) reported in Science, on the 'Mechanics of freezing in living cells and tissues'. He wrote, "*The single most important and fundamental concept in biological freezing is that regardless of the mysterious complexity of the biological matrix, freezing represents nothing more than the removal of pure water from solution and its isolation into biologically inert foreign bodies, the ice crystals.*"

It has been demonstrated that with slow freezing, ice crystal formation is generally confined to the extracellular spaces. However, tissue cells once frozen, may upon thawing and refreezing demonstrate uniform crystallization intracellularly, with formation of large destructive crystals of ice. The result often is lethal. This may account for the disastrous freeze-thaw-refreeze injury seen clinically, in which after initial extraction of cellular water, with increased permeability and trauma to cell membranes or endothelial lining of small vessels, a second freeze will affect intracellular supercooled water, resulting in the destruction of the cells (44,45,51,43,23).

Love, in outstanding explanatory fashion, furthered the concept of freezing phenomena in his chapter of Meryman's text (47). He points out that Koonz and Ramsbottom (1939) working with poultry, proposed that tissue initially froze extracellularly because the freezing point of lymph (equated with extracellular fluid) was higher than the cellular interior. Love

concluded that ice first forms outside the cells and is then augmented by intracellular water which diffuses through the cell wall and condenses on the ice surface because of the high osmotic pressure of the extracellular solution which has been concentrated by freezing. The temperature of the intracellular fluid never falls below its freezing point, since intracellular water is continuously being lost with a corresponding continuous reduction of freezing point.

Karow and Webb (28) further note that water cooled below 0 degrees C. does not crystallize until a temperature is reached that will permit the utilization of substances within the water to act as a nucleus for ice formation. The nuclear material may be relatively large inclusion bodies, such as colloids, it may be dissolved substances, or it may simply be water molecules clumped together by hydrogen bonds, called microcrystals. These authors report on Mazur's work in this same article, indicating that in slow freezing there is a tendency for water in cells to supercool, as there is a low probability that such a minute volume would contain a nucleation center. At relatively high temperatures (-10 degrees C.), extracellular water freezes. As water freezes in the external medium, its vapor pressure drops below that of the still supercooled intracellular water and thus draws free water from the cells.

It is pertinent here to define supercooling as the cooling of a substance below the temperature at which a change of state would ordinarily take place, without such a change occurring. For example, the cooling of a liquid below its freezing point without freezing taking place. This results in a "metastable state," defined as an excited stationary energy state whose lifetime is unusually long. A further example of the state of supercooling is provided by Burton (11), reporting on the observations of Sir Thomas Lewis in the British Medical Journal

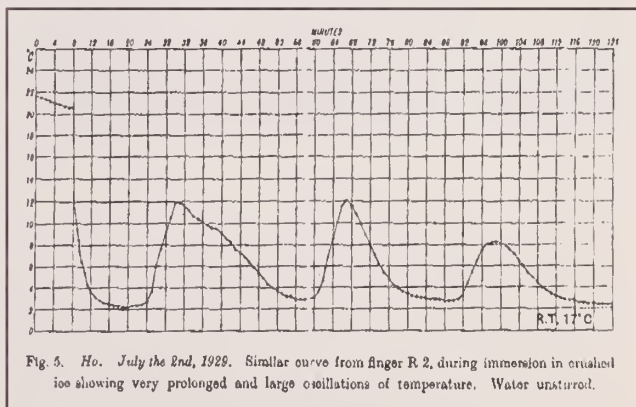


Fig. 5. From Lewis, T. Observations Upon the Reactions of the Vessels of the Human Skin to Cold, Heart 15 May 1930 p. 183. "Temperature curve during cold water immersion, demonstrating the 'rise and fall' response, gradually decreasing, the 'hunting' response of Lewis."



1941. "Observations on Some Normal and Injurious Effects of Cold upon the Skin and Underlying Tissues," wherein Lewis states that the freezing point of skin is about -1 to -2 degrees C., but that supercooling often occurs during which freezing is not demonstrated until the surface temperature is lowered from -5 degrees C. to -10 degrees C. Lewis further notes that the fat content of the skin affected supercooling. These concepts permit us then to define frostbite as true tissue freezing that occurs when there is sufficient heat lost in the cooling area to allow ice crystals to form in the extracellular spaces in slow freezing (the usual human freezing event) and to extract cellular water (51).

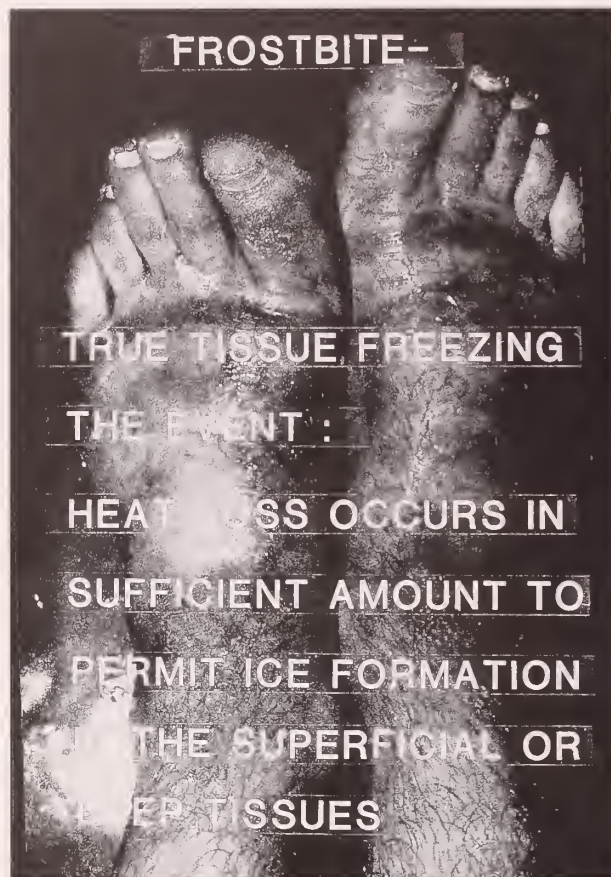


Fig. 6.

Pertinent literature review, in chronological order, can begin with the study of skin cooling by Sir Thomas Lewis in 1930, with his demonstration of rhythmic vasoconstriction and vasodilatation of cutaneous vessels, and his description of the "hunting" phenomenon (40). This finding was followed by the data of Grant, 1931, who demonstrated that cold dilatation was due also to increased flow through arteriovenous anastomoses (19). Harkins, 1977, suggested that a factor in frost gangrene was compression of vessels by local swelling and recalled the treatment of Bundschuh and Witek for

compression by multiple incisions (24). This method of relieving compartment compression pressure by escharotomy and fasciotomy was reported by Mills, 1973, and Franz et al., 1978 (52,18).

Blackwood in 1943, and Denny-Brown in 1945, who investigated WW II cold problems, demonstrated microscopic degenerative histologic changes in nerve and muscle in immersion injury as a direct action of cooling (6,15). Sayen in 1962 similarly reported the histologic changes found after immersion injury and noted that in extended cooling, tissue destruction was more severe, recovery more prolonged, with vacuolization and fragmentation of nerve axons (71).

Lange (33,34), 1945, Quintanella (67), 1947, Crismon and Fuhrman (13,17), 1947, utilizing rabbit ear chambers, demonstrated that after immediate thawing, ear tissue appeared normal, the circulation returned, followed by hyperemia, then massive edema, circulatory slowing and red cell clumping in the capillaries. Shumacher and coworkers (72,73,37,16,74,75,77), 1947-1960, in beautifully carried out laboratory experiments, developed a lasting bridge between the clinician and physiologist. They investigated the pattern of vascular change and injury following cold insult, and investigated varied drug therapy, in an attempt to improve circulation after thawing.

Kreyberg in 1949, in one of many outstanding articles, entered the controversy as to whether actual freezing of tissue is the lethal event. He considered that low temperatures, whether freezing or not, damaged cells and tissues, but that freezing was not as lethal as heat coagulation. He postulated that after the freeze, further tissue damage resulted during the period of thawing, demonstrated by hyperemia and stasis (32).

Scow (76) in 1949 reviewed the direct effect of cold on tissues of newborn rats, demonstrating remarkable distortion and retardation of growth in limbs and tails. He found cartilage cells to be susceptible to even brief refrigeration and concluded that changes occurred as the result of necrosis of cartilage cells normally active in skeletal growth. He considered his findings to support the hypothesis that the lethal effects of cold act directly on these cells, altering protein in the cytoplasm and nucleus. His pioneer work in growth cartilage freezing was followed by the further investigations of Bigelow (5), 1963, and Hakstain (20), 1972, who demonstrated that cold can destroy the cartilage of the epiphyseal plate which may result in digital shortening or angulation deformity of the digits, or joint dysfunction. Lytic destruction of periarticular cartilage and bone have been reported in adults and children (52,58). Lewis, 1951, similarly demonstrated that degenerative changes in muscle occurred almost immediately after exposure to freezing (38). He later, 1953, studied the effects of Rutin, a flavinol glucoside, and hydergine, a

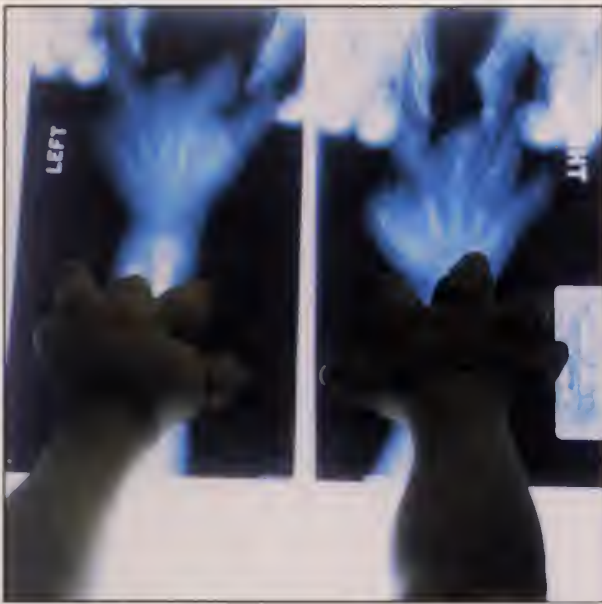


Fig. 7. Photograph of hands, and AP views of hand and wrist, bilateral, of an eight-year-old child. At age four and a half months, she had been left in the snow at minus thirty-seven degrees F. (-38 deg.C). The baby had a core temperature less than 70 degrees.F, and sustained severe freezing of hands and feet. She was successfully warmed in water. She suffered severe hypermobility of joints, intrinsic muscle degeneration. The x-ray reveals absence of the carpals, and digital epiphyseal necrosis, with phalangeal shortening. An arthrogram of the wrists demonstrated total absence of cartilage as well as bone.

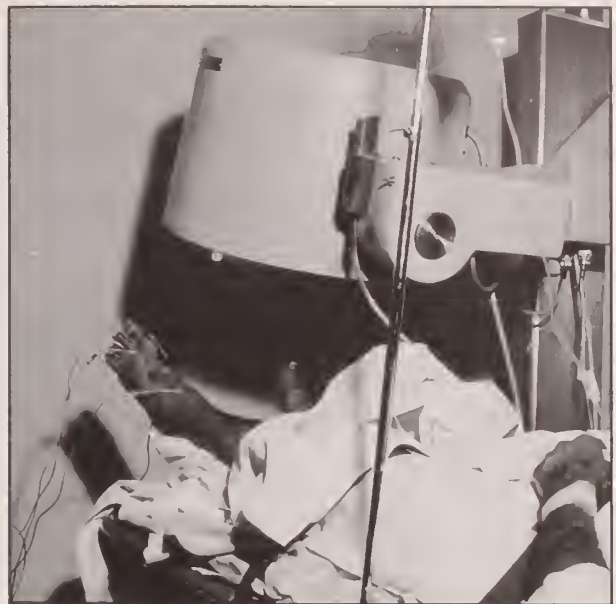


Fig. 8. Photograph of patient with history of operation for accessory pedal digits, with evidence of post operative vascular arterial deficiency, confirmed by arteriogram. Here the patient is undergoing deep temperature thermistor evaluation and simultaneous evaluation of blood flow utilizing a Gamma camera and technitium 99m. Providence Hospital, November 1971.

dihydrogenated ergot vasodilator, to determine their effects on experimental cold injury (39). Shumacher, 1951, had also studied the effect of Rutin, demonstrating more beneficial effects in reducing gangrene than Lewis (74). Of interest is that both Rutin and hydergine are now considered efficacious in overcoming the effect of free radical injury -- now considered a likely cause of cell damage after thawing and reperfusion of the vascular system (65,21,14,1,87,50,10,42).

In keeping with the military interest in cold injury, Orr, 1953, reported on the findings of his cold injury research teams, in Japan and Korea, during the Korean war. One member of that team, H. T. Meryman, a Naval medical officer, was to go on to become one of the world's leading investigators in cold-related science and cryobiology (44-49). His contributions include the role of extracellular ice formation and cell damage, the effect of extracellular solute concentration and the methods of ice crystal nucleation. He has presented a lucid explanation of the effect of the rate of freezing (slow, rapid) in cellular biological systems (44) 1955, (45) 1956, (46) 1957. His work includes the development of injury classification, the postulation of a time-temperature relation in clinical cold injury, and the presentation of a pattern of ice crystal formation with the resulting biochemical effects. Meryman summarized to that date, the mechanism of slow freezing injury in tissue, namely,

extracellular ice crystals which displace and partially dehydrate soft tissue cells. While some tissues may be injured or destroyed by this process alone, most tissues passively collapse without significant mechanical injury. However, the removal of water results in high concentrations of electrolyte and other cell constituents. This concentration, through biochemical means, produces a cumulative injury to the cell. As the temperature falls from the freezing point to between -10 degrees C. and -15 degrees C. more water is frozen out, increasing the solute concentration and the potential for injury.

Bellman and Adams-Ray, 1956, and Sullivan and Towle, 1957, investigated vascular response to cold, finding that cold trauma involving rapid freezing, then rapid thawing, injured the tissue less than slow thawing (4). Other studies indicated that in the post thaw stage there was an increase in the rate of blood flow, accompanied by the emergence of many platelet emboli from the area of injury. Stasis was found to begin in the venules, spreading throughout the vascular bed. Hemoconcentration was considered the cause of stasis following local cold injury (78).

In 1960, Mills, Whaley and Fish (51) reviewed their early clinical results of rapid rewarming for local cold injury and proposed for the first time that the condition be treated with a protocol of care. This total system approach included the avoidance of trauma to the frozen extremity, rapid rewarming in warm water



(preferably in a whirlpool bath) at 42-48 degrees C. later changed to 38-42 degrees C. (52). Post thaw whirlpool at 32-37 degrees C. was utilized to massage tissue, promote circulation and dilute the superficial accumulation of bacteria and discourage infection. Isotope and enzyme studies were utilized to determine circulatory status, and for early diagnosis of extent of injury. Recognition of the freeze-thaw-refreeze injury was noted, the latter quite possibly a result of intracellular ice formation as a result of the second freeze, and therefore, usually lethal (51-54,57,58). In that paper (51) the authors suggested that the time honored description of first-degree, second-degree, third-degree, fourth-degree frostbite be changed to a more descriptive clinical diagnosis of superficial (first degree to second degree) and deep (third degree to fourth degree).

Luyet, one of cryobiology's pioneers (41), 1964, published observations on the invasion of living tissue by ice, listing three stages of invasion: 1) superficial freezing; 2) intercellular freezing (extracellular spaces); and, 3) intracellular freezing. He demonstrated shrinkage of erythrocytes by osmotic differential in extracellular freezing.

Mundth, also in 1964, (60, 61) demonstrated platelet clumping soon after thawing, arising from injured endothelium of vessel walls followed by corpuscular aggregation that eventually became occlusive. Mundth recognized that local tissue injury from freezing was associated with local vascular damage after thawing involving increased endothelial permeability, intravascular cellular aggregation, capillary stasis, occlusion of small vessels by cellular aggregates, and thrombosis. He demonstrated that low molecular weight Dextran (m.w. 41,000) given intravenously prior to freezing, improved tissue survival after freezing by improving capillary flow, and inhibition of corpuscular aggregation. This work was corroborated by Anderson and Hardenbergh. 1965, but only when test animals were rapidly thawed after freezing (2).

In a scholarly paper by Karow and Webb, 1965, in the second volume of Cryobiology, the newly created forum for cold studies, a 'Theory for Injury and Survival' was presented (28). This concept was based on the assumption that bound water, in the form of lattices, was essential to cell integrity, especially protein structure and function. Death in freezing seemed to occur primarily as a result of the extraction of bound water from vital cellular structures. This extracted water, incorporated into growing ice crystals, left proteins, dehydrated and denatured.

His explanation of why extracellular freezing initially began, was that water cooled below 0 degrees C. does not crystallize until a temperature is reached that will permit the utilization of substances within the water to act as a center or nucleus for ice formation. That

nuclear material may be relatively large inclusion bodies, such as colloids, it may be dissolved substances, or it may be simply water molecules clumped together by hydrogen bonds, the microcrystals.

Hanson and Goldman, 1969, reviewed the etiology of cold injury, with particular reference to injuries of WW I, WW II and Korea, deciding that after review of all data, prediction of the incidence of cold injury was almost impossible. In 1969, Hardenbergh and Ramsbottom (23) confirmed the findings of Mills (51) that "double freeze" injury was indeed more clinically significant, causing much more harm than a single freeze.

Knize et al., 1969, proposed a system for clinical prognosis or tissue loss after frostbite, based upon duration and condition of exposure, and the level of lowest temperature reached (31). Sumner et al. (80), 1970, in similar fashion developed a prognostic sign based on dog experimentation that indicated that blood flow in the involved extremity twenty-four (24) hours after freezing had prognostic significance. Xenon 133 was used to predict the extent of tissue loss in frostbite as early as ten minutes post thaw. Mills (51,52,58) and Salini (69) in 1986, reported similar prognostic use of the radio isotope Technetium 99m pyrophosphate.

In 1971, Meryman (48) followed his other investigations with a comprehensive review of major theories regarding injury following freezing and thawing, and the mechanism by which the osmotic loss of cellular water might produce injury. He suggested that hypertonic alteration of cell membranes is preceded by a stress which increases as osmolality is increased. His contention is that reduction in cell volume leads to membrane injury.

In 1974, Meryman (49) followed that study with a scholarly dissertation on freezing injury and its prevention in living cells, with particular emphasis on the role of the ice crystals and the mechanism of freezing injury through solute concentration. Meryman considered salt concentration as a cause of injury when salt denaturation of membrane components occurred. Evidence was presented indicating the primary site of cell injury is the cell plasma membrane, which includes membrane permeability alterations, along with the effect of elevated extracellular osmolality resulting in loss of cell water and cell volume reduction.

Mazur (43) in 1970, reviewed the responses of living cells to ice formation and considered that although the freezing point of cytoplasm is usually above -10 degrees C., cells generally remain unfrozen and therefore supercooled to -10 degrees C. or -15 degrees C., even when ice is present in the external medium. This indicated that the cell membrane can prevent the growth of external ice in the supercooled interior and further suggests that cells neither are, nor contain, effective nucleators of supercooled water. Mazur believed that to understand

the solution effect as a mechanism of cell damage, one need consider that four discrete events occur during freezing: 1) water is removed as ice; 2) solutes of high and low molecular weight concentrate; 3) cell volume decreased; and, 4) solutes precipitate. Contrary to the theories of Lovelock and Meryman (47), Mazur considered the cause of injury from extracellular ice to be that it exerts sufficient force to rupture plasma membranes, or the membranes of organelles such as mitochondria. His rationale for this suggestion is that recrystallizing ice crystals can disrupt protein gels, and that cells killed by intracellular freezing have suffered membrane damage and become leaky.

Carpenter et al. (12) in 1971, demonstrated the beneficial effects of rapid rewarming at 42 degrees C., showing that by that method endothelial cells remained attached to the arterial intima, the internal elastic lamina remained intact with the media less distorted. During slow thawing the endothelial cells were almost completely shed into the vascular lumen, the internal elastic membrane was disrupted and cells of the media distorted and necrotic.

Molnar et al. (59) in 1972, attempted an analysis of events leading to freezing, using finger temperatures. It was concluded that the incidence of either freezing or cold induced vasodilation could not be correlated with the relative cooling rate because of indeterminate supercooling. The authors concluded that the factors which induced crystallization on the one hand and vasodilatation on the other remained to be discovered.

In 1973, and later, Mills (52,54,55,57,58) described fasciotomy as a method of relieving the lethal tissue effects of increased compartment space pressure. This mode of decompression was further investigated by Franz (18) in 1978, on laboratory dogs, demonstrating its effectiveness.



**Fig. 9.** C-1098-88

Young man, age 18, found with multiple injuries, hypothermia (86 degrees F, 0 - 30 degrees C.) and severe freezing injury of hands and feet in Arctic Alaska. Exposure unknown, ambient temperature <-5 degrees F. (-15 degrees C.) Rewarmed by warm intravenous fluid and warm moist oxygen until core temperature was 97 degrees, then warm water warming of hands and feet. Transferred to Anchorage with insensate feet, severe edema, compartment pressure measurements 60-80mm Hg throughout feet. Fasciotomy performed at 24 hours, bilateral feet.



**Fig. 10.** C-1098-88.

Multiple fasciotomy incisions both feet, medial and lateral, toe to mid-calf. Immediate color change, cyanosis to pink, extrusion of muscles and evidence of edematous neurovascular bundles with venous clotting, but almost immediate return of arterial pulses in posterior tibial medial and lateral plantar arteries. Fasciotomy extended distally until all tissues were mobile.



**Fig. 11.** C-1098-88

Soon after fasciotomy, demonstrating increased perfusion and tissue expansion. Fasciotomy extended further in 8 hours, into tarsal tunnel with further increase in perfusion.



**Fig. 12.**

The wound was closed by mesh graft on the 10th day with some of the incisions loosely closed by continuous tension suture, decreasing the area to be grafted. The tissue loss was limited to the distal toe tips and distal phalangeal tufts.

Bowers in 1973, investigating in vivo freezing, viewed ultrastructural changes occurring in capillary endothelium by electron microscopy. He discovered no precipitous changes in muscle cell mitochondria or capillary endothelium as a result of hypoxia after cooling tissues



at 2 degrees C. or supercooling to -13 degrees C. However, reducing the temperature by 1 degree C. per minute until freezing occurred, and continuing to cool for ten minutes, followed by rapid rewarming, resulted in consistent mitochondrial damage in muscle cells. There was also marked degeneration of associated capillaries (9).

In 1980, Vanore (85), followed by Purdue (66) in 1986, and Britt (10) in 1991, conceived, along with their colleagues, well detailed summaries of the events leading to the tissue effects of cold injury. The events following cold insult were categorized using the direct effect of the cooling and cold period, the freezing period, and the immediate and delayed post thaw periods. Their reviews are comprehensive, and rich in the pertinent bibliography of cold injury. Their method of event analysis will be utilized in the summary of this paper.

Beginning in 1981, Robson, Heggers, McCauley, Phillips et al. (68,62,25-27) published observations on metabolites of arachidonic acid (PgF2 alpha and TxB2) in frostbite blister fluids. The authors have suggested these metabolites as a cause of dermal ischemia, and a possible cause of the progressive vascular changes seen in cold injury. Based upon this data, the group developed a 'rational approach' to treatment of frostbite based on the pathophysiology of freezing injury. Having demonstrated the breakdown products of arachidonic acid, the authors used anti-prostaglandin agents, and thromboxane inhibitors to preserve the dermal microcirculation. In 1983, Heggers and Robson (25), published an erudite summary of the phenomena found in the regional vasculature after thawing. At the moment, the therapy has limitations in the area of associated trauma, or refreezing injury (58).

Marzella in 1989, utilizing light and electron microscopy, studied morphologic changes in vascular endothelium of the skin (42). He concluded that the endothelial cell is the initial target of the injury induced by freezing, and concluded further that the injury is mediated by a non-free-radical mechanism. The statement is made that "by now it is generally agreed that direct thermal injury alone is not sufficient to cause cell death." He suggests that the initial freezing impairs microvascular function, leading to edema, stasis, thrombosis and finally ischemic necrosis. This may be followed by the production of arachidonic acid metabolites after thawing. These result in inflammatory responses that modulate vascular contraction and permeability, platelet aggregation and recruitment, and activation of leukocytes. He brought a "new player" into the game, indicating the participation of free radicals in the induction of tissue damage. Marzella pointed out that the consideration of free radical injury has been suggested by evidence showing that superoxide dismutase (SOD), an oxygen radical scavenger, and iron chelates given at the time of thawing protect against

frostbite, and that physiologic and biochemical evidence from other experimental systems suggest that endothelial cells are susceptible to free radical mediated injury.

However, in Marzella's rabbits freezing caused an immediate separation of endothelial cells from the internal elastic lamina. It was considered that the separation was present even in samples removed immediately after freezing and before skin thawing, so that reperfusion could not be considered responsible for that lesion. Separation of endothelial cell junctions was seen in venules and capillaries soon after freezing. It was also suggested that other inflammatory mediators released after injury such as leukotrienes, may have contributed to separation of cell junctions.

From the foregoing material, we can list the course of events in freezing-thawing stages -- of freezing injury.

#### A. Cooling: Supercooling: Freezing Stage

1. First, with exposure to cold, there is an early tissue response to cooling. This is described as a cold-induced vasoconstriction (CIVC) followed by a cold induced vasodilatation (CIVD), the "Hunting Response" (Lewis) (40).
2. Another hypothesis states that at the same time or as cooling continues, arteriovenous anastomoses develop with shunting of blood distally (Vangaard) (84).
3. Soon, after sufficient heat loss occurs to allow freezing, ice crystal formation in the extracellular fluid spaces occurs, with extracellular freezing (Meryman) (45). This event is precipitated by inclusion bodies, and microcrystals. It is possible that some structural damage may result from continued ice crystal growth (Karow, Webb) (28).
4. Extracellular osmotic pressure increases (Meryman) (48) resulting in cell volume reduction and solute concentration in the extracellular spaces and interstitium.
5. As freezing continues, there is an elevated concentration of electrolytes protein denaturation, inter and extra cellular pH changes, inter and extra cellular dehydration freezing of extra cellular water, loss of protein bound water in the cells, and destruction of essential enzymes (Mazur) (43).
6. As cooling and freezing continues, cell membrane damage occurs with impairment of microvascular function and increased cell wall permeability, with critical endothelial cell injury, and endothelial separation from the internal elastic lamina of the arterial wall

(Marzella) (42). At this time, severe injury to chondrocytes may occur since cartilage, particularly epiphyseal cartilage, is susceptible to freezing damage.

7. Further insult causes ultrastructural capillary damage, mitochondrial loss in muscle cells, and injury to other intracellular structures (Lewis) (38).

#### B. Vascular Stage -- Thawing (Rewarming) and Post-Thaw Stage

1. Depending upon the method of thawing, post thaw hyperemia, ischemia, cyanosis, even total circulatory failure usually develops (Bourne) (7), (Grant) (19), (Quintantella) (67).
2. Proximal blebs, distal blebs, or no blebs appear. The usual event is that of vasodilatation, edema, and stasis (Meryman) (48), (Shumacher) (75).
3. Corpuscular aggregation begins with thawing, often associated with progressive ischemia, or with hyaline plugs in the vascular tree (Mundth) (60), (Bourne) (7), (Marzella) (42). Occasionally, because of associated or combined injury increased pressures may develop in soft tissue compartments (Mills) (54,55,57).
4. Changes related to reperfusion injury, with formation of oxygen free radicals, neutrophil activation and other inflammatory events (Ward) (87).
5. An early response in the thawing stage, and perhaps in cooling too, is the arachadonic acid cascade, liberating prostoglandins and thromboxane predisposing to vascular clotting (McCauley) (62), (Robson) (68).
6. Production of proteolytic enzymes with increased membrane permeability.
7. Eventually a) vascular reconstitution and clot dissolution begins, or b) capillary and peripheral vessel collapse occurs, followed by micro and macro vascular thrombosis, venule and arterial obstruction by thrombosis, tissue ischemia, necrosis, and gangrene, resulting in loss of the affected part or area.
8. Following thawing, should refreezing occur, intracellular ice formation is most probable, resulting in cell and vital organ destruction. Thawing in this usually deep injury, results in unrelieved thrombosis, stasis, failure of cell repair, loss usually near or at the level of the second freeze (Hardenberg) (23), (Meryman) (45), (Mills) (51).

#### SUMMATION: The Milieu of Environmental Freezing:

Despite all we have listed as causative elements in sequence of freezing, little has been said of the other factors so important in determining the final result in the human. These variables are in the realm of weather, inadvertent accident and trauma, and individual human physiology and anatomy, often difficult to anticipate, measure or predict.

The state of health and physical condition of the victim are also vital factors, that include associated factors of alcohol and drug use, and mental state at the time of exposure.

The individual's neurovascular integrity in peripheral areas is a major factor if disease states such as diabetes, arteriosclerosis, vasculitis, labile vasomotor disturbances and Raynaud's phenomenon or Buerger's Disease are present.

Associated trauma, preceding freezing, such as extremity strain, sprain or fracture pose major problems, as does the presence of a penetrating wound, blunt trauma, or blood loss from any cause. Freeze injury is further influenced by the degree of hypovolemia or dehydration present, causing further distal vascular deficiency prior to the onset of freezing.

Little considered are problems of rescue and survival, often resulting in refreeze injury, and perhaps the irreparable trauma occurring at the junction of frozen-nonfrozen tissue as 'brittle' tissue segments are stressed when the cold victim must walk to survive a wilderness catastrophe.

To emphasize the freezing patterns facing rescuer and attending medical personnel, Mills and Pozos (57) and Mills (58) in 1991, listed seven modes of freezing injury found in Alaska: 1) true frostbite, superficial or deep; 2) a mixed injury, immersion (wet cold) injury followed by freezing which is usually disastrous, with great tissue loss; 3) freezing, then thawing at any temperature followed by refreezing, again a disastrous event, with total tissue destruction, and early mummification of distal tissues, usually occurring in five to seven days; 4) hypoxia, high altitude environmental injury, usually associated with hypovolemia and dehydration, and extremity freezing. The prognosis is poor if associated with other trauma; 5) extremity compartment compression from any cause, followed by freezing. Very poor results followed if the compartment pressure is not relieved by medical or surgical means; 6) extremity fracture or dislocation, followed by freezing. The results are poor if the fracture dislocation is left unreduced, and the best results follow rapid rewarming; 7) hypothermia, associated with freezing injury to the extremities.



## RAPID REWARMING WARM WATER BATH (108°F) (42°C)



Fig. 13. C55-60. Rapid Rewarming Warm Water Bath (108 degrees F.) (42 degrees C).

**Frame #1:** The patient sustained freezing of hands and feet on the Arctic Slope when marooned in the open as a result of a vehicle accident. Winds were 80 knots, ambient temperature between -20 degrees C. and -26 degrees C. The patient lost his overboots and gloves in the accident. His entire exposure time he states was 15 to 20 minutes, followed by 45 minutes in the wrecked vehicle awaiting rescue. Upon rescue, he was warmed in water at 160 degrees F. (42 degrees C.) - this warming and care directed by radio from Anchorage. The patient was then transferred from the Arctic Ocean shore to Anchorage by air travel at 24 hours. On arrival, the hands demonstrated large, clear, pink blebs extending to the finger tips - these being excellent prognostic signs, especially the fact that the blebs are distal in position, extending to the nailbeds. Note: Only after rapid rewarming in warm water is there return of sensation in the finger tips, this remaining until blebs appear in the dermis and epidermis and separate those tissues from the deep structures.

**Frame #2:** Constant twice daily whirlpool is prescribed with digital exercises, using surgical soaps pHisoHex or Hibiclens or Betadine.

**Frame #3:** By the third week, epidermal eschar has formed preventing joint motion.

**Frame #4:** This eschar, periodically when the tissue permits, is incised to allow joint motion. This procedure (escharotomy) usually is performed from the 14th to the 31st day.

**Frame #5:** Digital exercises are done at frequent intervals, at least four times daily, associated with whirlpool and biofeedback training. By this time, the fifth week, there is loss of volar fat pad, loss of nails, resolving hypesthesia.

**Frame #6:** The anatomical result is good, but volar fat pad loss and intrinsic muscle loss is obvious. The patient has considerable atrophy as demonstrated in frame #6 of the first dorsal interosseous, and this is true of the abductor digiti quinti.

In addition, more recently (1993), to that list has been added: 8) freezing superimposed on small vessel disease (as found in diabetes); 9) freezing injury in children, with epiphyseal necrosis; 10) congenital deformity with superimposed freezing; 11) frostbite with superimposed burns or burn injury with superimposed frostbite.

Having demonstrated clinically the 11 freezing patterns seen in Alaska, one finds the picture confused by thawing methods. Often, long before the victim of freezing is seen in the hospital, some form of thawing,

and occasionally refreezing has occurred, regardless of the method of thawing. We can recognize generally four methods of thawing, as seen after arrival in the emergency area: 1) rapid rewarming in warm water (37-41 degrees C.); 2) gradual (spontaneous) thawing at room, cabin or tent temperature, or in a sleeping bag, so that the thawing range varies; 3) delayed thawing, utilizing ice, ice water, or snowpacks often accompanied by friction massage; 4) thawing by excessive heat, that has included car heater, diesel generator exhaust, oven heat, hot water, camp fire, or any heat greater than 48 degrees

C-807-82

## BAREFOOTED SNOW TRAVELER-REFREEZE INJ.



Fig. 14. C-807-82. Barefooted Snow Traveler - Refreeze Injury

**Frame #1:** This patient demonstrating psychiatric behavior, had multiple episodes of 'walking in the snow' without shoes over at least seven to ten days. He was rescued, revealing poor nutrition and at the time he was found had mummification of the toe tips, severe edema and pallor of feet with a history of previous warming in warm water elsewhere. The pulses at the time were absent. The feet were cold, were totally insensitive; and it was considered that he had had freeze-thaw-refreeze injury on numerous occasions.

**Frame #2:** Mummification of the toe tips is present by day #10 as seen on the arrival in the emergency room. Plantar pads and heels are involved as well. Doppler pulses were absent at the level of the malleoli bilaterally.

**Frame #3:** Digital subtraction lower extremity angiogram performed by way of the right brachial vein and anterior vena cava, demonstrate occlusion of all major vessels 7 to 8 cm above the ankle joint.

**Frame #4 and 5:** These frames demonstrate necrosis, dry gangrene and separation in the supramalleolar area by the 17th day. The isotope studies technetium 99m demonstrate total loss of perfusion of both feet. The patient, despite injuries and necrosis and toxicity, had refused amputation of feet.

**Frame #6:** After development of increasing toxicity and the onset of increasing sepsis, a court order was obtained for patient care, and a modified guillotine amputation was performed on the 19th day or 13th post-admission day. Twelve days later, split thickness skin graft was applied for wound closure. One month later, the revision below knee amputation was performed bilaterally. Experience has dictated that in severe cold injury, the best results are obtained by guillotine and modified guillotine amputation to rid the patient of the necrotic, gangrenous and infected tissues. After stump granulation, amputation revision will allow adequate clean amputation site closure.

C., often 65-90 degrees C., that causes burning of the frozen part. Multiple thawing methods are utilized by the victims or rescuers, including combinations of all four above. Not all are appropriate, methods one and two are least harmful. Unfortunately, less than 20 percent of patients arrive in the ER with extremities still frozen.

## WHAT IS THE FUTURE?

We have in this paper, taken a walk with the physiologist, basic scientist, and clinician, through the years

of the development of the understanding of the pathophysiology of freezing injury and associated cooling. Lurking on the horizon is the likely involvement, in hypothermia and freezing, of free radical formation as a result of organ, full body or extremity reperfusion. Miller (50) in 1978, considered that irreversible damage is related to the oxidation of protein sulfhydryl groups, to intermolecular disulfide bonds. This oxidation process probably involves hydroxyl radicals. It was suggested that removal of the -OH radical by a scavenger would add cryoprotection to cell membranes. Classic



C-761-82

## HELICOPTER ACCIDENT--MULTIPLE TRAUMA



**Fig. 15a.** C-761-82 #1. Helicopter Accident - Multiple Trauma.

**Frame #1:** In the high Arctic, Bering Sea Island, the patient's helicopter crashed into a ridge. The helicopter had been buffeted by 50 mile per hour winds, the temperature was -8 degrees F. (-22 degrees C.). The patient was the lone survivor, his companions dying of injury and exposure. The victim was without gloves at crash time, but had adequate boots. His right arm was fractured. Upon regaining consciousness, his hands were found to be frozen. He was able to crawl and move about, discovering he had multiple injuries restricting motion. He did, however, crawl to a high ridge above the crash site on numerous occasions over a 36-hour period to signal for help with a locator transponder. During this time he sustained multiple freeze injuries to his left hand and attempted to protect from further freezing his right hand. He was rescued by snowmachine, then flown to Anchorage. Spontaneous thawing occurred in transit. Upon arrival at the hospital, his examination revealed him to have (1) bilateral freezing injury to the hands; (2) a Monteggia fracture dislocation, right; (3) a closed head injury; (4) compression fractures of two thoracic vertebrae; and (5) soft tissue injuries of the neck and question fracture compression of several cervical bodies, minimal. The initial admission photograph, despite thawing, demonstrates very small blebs which are primarily proximal in position. The tips of the fingers were very cyanotic. Both hands, though insensitive, seemed to have adequate warmth.

**Frame #2:** X-ray examination of the right elbow revealed a dislocation of the radial head and fracture of the proximal ulna (a Monteggia fracture).

**Frame #3:** The day after admission, the fracture of the ulna was openly reduced and gently fixed with a Rush intramedullary nail. The radial head spontaneously reduced itself when the ulna was fixed. The procedure was done to facilitate motion, care and physiotherapy and reduced swelling that might limit vascular supply.

**Frame #4:** Prior to surgery, a technetium scan demonstrated no perfusion of the fingers beyond the metacarpophalangeal junction of the right hand, and on the left there is capillary perfusion of the proximal and midphalanx of the fifth finger and proximal phalanx of the fourth and third finger and only a small portion of proximal phalanx of the thumb and index finger.

**Frame #5:** A week after the accident, the left hand demonstrates advanced mummification of the distal digits of the left hand, dry with edema and early liquefaction necrosis of the digits of the right hand. The pattern of the left hand is a typical freeze, thaw, refreeze injury. The pattern on the right may represent the effect of unreduced fracture dislocation, over a 36-hour period and the uncertain result of spontaneous thawing, particularly with fractures or dislocations.

cryoprotective agents, dimethyl sulfoxide and glycerol are hydroxyl radical scavengers. Later studies have investigated reperfusion injury and the involvement of oxygen free radicals, and activated neutrophils incriminated in endothelial injury during reperfusion (1,14,21,65,87). These areas are considered likely fields

for investigation. Much like any tissue in an ischemic state (e.g. myocardium in heart disease and surgery), freezing and hypothermia may be considered a reperfusion problem and subject to free radical injury.

If the new players in the game, the oxygen free-radical, the activated neutrophil and other inflammatory

C-751-82

# MULTIPLE TRAUMA, REFREEZE - SPONT. THAW

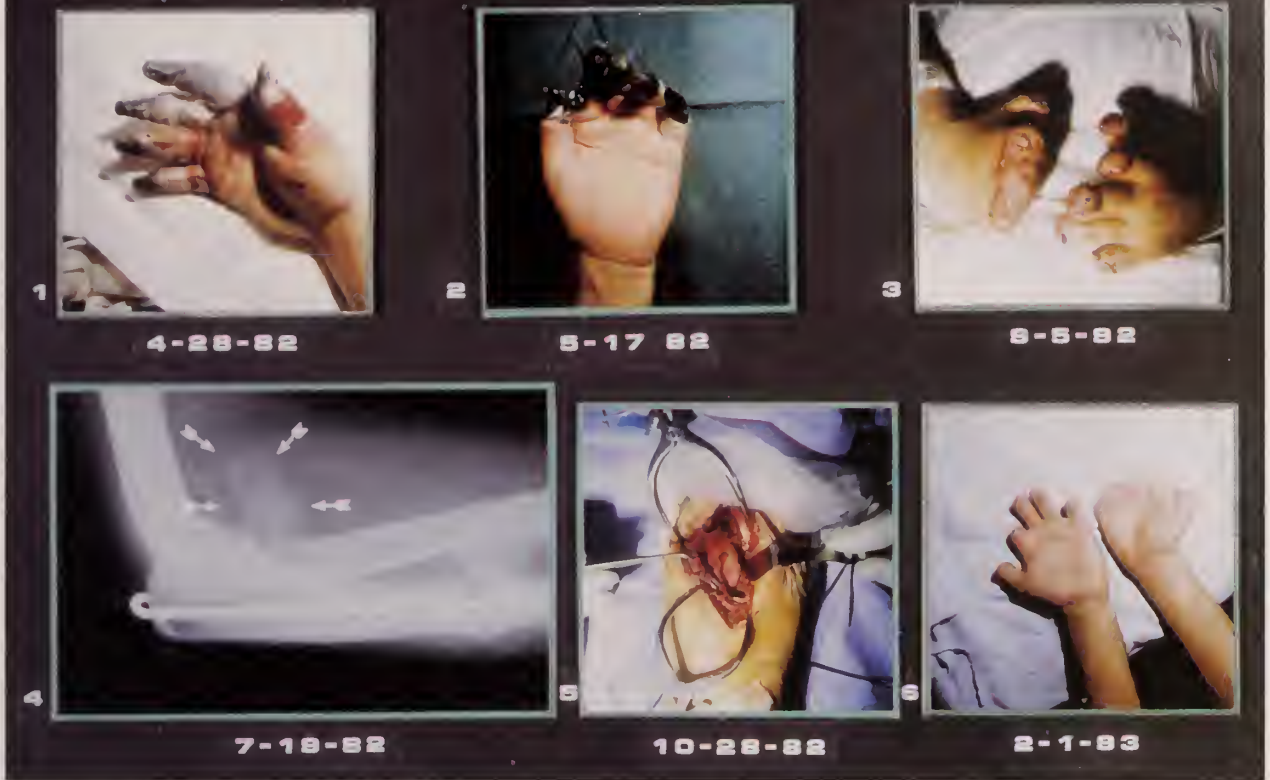


Fig. 15b. C-761-82 #2.

**Frame #1:** After the surgical reduction of the Monteggia fracture, the postoperative technetium 99m examination demonstrated a partial return of capillary perfusion to both hands. However, on the right hand, by the end of two weeks, severe necrosis, mummification, infection and the edematous reddish-orange discoloration of tissue necrosis and vascular failure is present.

**Frame #2:** At 30 days, the dry, mummified tissues demarcating anatomically are present, and the hand is ready for surgical debridement.

**Frame #3:** Eventually, guillotine modified, amputation of the metacarpophalangeal level on the right and at varied phalangeal levels on the left was performed - this at the end of four weeks. At six weeks, further debridement and split thickness skin cover of both hands was performed.

**Frame #4:** Two months post-injury, an x-ray of the elbow revealed a soft tissue calcification extending anteriorly from the radial head region. This was mushroom-shaped, measuring approximately 3 by 3.5 cm, somewhat ovoid. It appeared to represent either a myositis ossificans or calcified hematoma. A radial-ulnar synostosis was present at the ulnar fracture site and site of operation.

**Frame #5:** Six months post-trauma, the right elbow was explored anteriorly. A large, pedunculated bony mass was removed. The mass was found to have lifted the radial nerve upward for a distance of 2 cm, and displaced the radial artery and soft tissues. The mass originated from the radius, just below the radial head in the area of the annular ligament. The radial-ulnar synostosis was removed, permitting supination of 80 degrees, a motion previously lost.

**Frame #6:** Ten months after injury, the patient has a 'paddle hand' on the right with a segment of thumb and phalanx remaining. Small remnants of the proximal phalanges remain as well. On the left, sufficient phalangeal residual is present for the patient to continue his work as an electronic engineer. A large toe transfer for a right thumb and web space releases are contemplated further on the right hand. The findings in this case represent the disastrous results of severe associated fracture followed by thawing other than rapid rewarming, in this case spontaneous thawing, and represent the need for immediate care and early reduction of the fractures or dislocations. Thrombolytic therapy was inappropriate in this case because of the combined injuries to the head and neck and thoracic spine, the use of thrombolytic enzymes considered to likely cause intracranial or intraspinal bleeding.

and immune responses give cause for future research as a cause of cold injury, we still must account for injury from 'old friends' not yet overcome. The almost universal basis of treatment is rapid rewarming, to rapidly melt extracellular ice crystals, avoiding the lesser damage of mechanical damage by ice, and the more likely damage of dehydration within the cell.

Unexplained, however, is the often unexpected and poor result obtained from rapid thawing — free radicals? Activated neutrophils? Deep freezing? Extended duration of freezing? Extremely low temperatures?

Since most cases presented to the emergency room have spontaneously thawed, perhaps we can seek a method of care improving that thawing result. This



## OPEN FRACTURE--FREEZE--RAPID REWARMING



Fig. 16a. C-ANFX. Open Fracture - Freeze - Rapid Rewarming

**Frame #1:** A trapper was injured, sustaining an open fracture of the distal radius and ulna on the left arm. He sustained freezing of the hand and wrist while seeking help. The warming at rescue was rapid rewarming in warm water, the forearm was splinted to protect the fracture.

**Frame #2:** Twenty-four hours after the warming, the blebs are large, light-colored, clear and extend to the finger tips, an excellent prognostic sign.

**Frame #3:** The open area was debrided after thawing with thorough irrigation of the wound, fracture repositioning, antibiotic coverage (Streptomycin and penicillin) in vogue at that time. The arm was splinted only, and digital exercise immediately done. The wound was packed open and allowed to granulate closed.

**Frame #4:** The displaced (compound) fracture, with displacement and skin penetration of the fragments of radius and ulna is demonstrated.

**Frame #5:** The forearm and upper arm is contained in a plastic open splint, allowing for whirlpool therapy, motion of the joints and observation is permitted without occlusive dressings present. Whirlpool with pHisoHex soap (Hibiclens, Betadine) is used for constant debridement twice daily.

**Frame #6:** Tissue healing was eventually sufficient to permit surgical fixation of the fragments. The patient sustained no anatomical loss. Fractures, followed by freezing, appear to do best if rapid rewarming is utilized as a thawing method. This can be supplemented if necessary by fasciotomy, stabilization of the fracture at least by splinting, and immediate reduction of fracture dislocations or dislocations.

would be even more appropriate in a military setting, particularly with beleaguered troops, in freezing weather and hostile terrain, for it is much easier to care for hundreds or thousands of frozen extremities by spontaneous thawing than rapid rewarming in water.

Perhaps further investigation may permit us better methods of avoidance of freezing, or barring that, provide drugs taken orally to prevent vascular clotting and endothelial injury. Further, there is need for a protocol to avoid the severe damage to tissue in freeze-thaw-refreeze injury, and also avoid epiphyseal necrosis in children's freezing injury.

Perhaps, as many authors in the past have suggested, the best avoidance of cold injury insult is a program of prevention, with improvement of clothing, gloves, boots and protective equipment. Again, however, it is likely that the cold problems will be with us forever. Rescue personnel and medical practitioners must be prepared for its diagnosis, evaluation and need for latest state of the art care -- for it isn't probable that the forces of nature, snow, ice, wind, violent storms at sea and perils of the mountains will change -- and even less probable that the interests, work and play habits, and health destroying actions of man will change much either.

## MOUNTAIN ACCIDENT AND RESCUE-MT. MCKINLEY



Fig. 16B. C-593-79 #1. Mountain Accident and Rescue - Mt. McKinley.

**Frame #1:** Late in May of 1979, a climbing party of three fell, roped together from a cornice of the West rib. Two died in the fall. The survivor was found hanging head down, a rope wrapped about his right knee and thigh. The red asterisk is the site of the University of Alaska research camp at 14,000 feet (4,267 meters), and the red arrows the area of the fall - a fall of approximately 2,000 to 3,000 feet (600 to 900 meters). The fall was observed from the camp at approximately 10 PM.

**Frame #2:** The medical research party reached the victims about six hours after the fall. The patient was carried back to 14,300 foot base camp and examined there by a climbing physician with another party.

**Frame #3:** He was placed in a tent where examination found him to be semicomatose and delirious. He had an obvious dislocation of the right knee with freezing of the hands and feet. The hands began to thaw in the tent so that rapid rewarming was carried out. The right leg was packed in ice to prevent thawing and because of the extent of the injury.

**Frame #4:** Helicopter rescue was carried out approximately 20 hours after the fall. Note: In helicopter rescue, it is important to prevent further freezing injury or refreeze injury to the patient by avoiding the exposure of the frozen parts to wind chill from helicopter rotors. Helicopter pilots often keep rotors turning at altitude to avoid danger of failure to restart engines.

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## MT. MCKINLEY - MULTIPLE TRAUMA AND COLD INJURY



Fig. 16c. C-593-79 #2. Mt. McKinley - Multiple Trauma and Cold Injury.

**Frames #1, 2, 3 and 4:** Upon admission, the patient was found to be semicomatose, had thawed both hands which were now edematous, cyanotic and still cool. X-ray examination demonstrated probable fracture of L2, and an angiogram eventually revealed an obstruction of the popliteal artery 3 cm above the knee joint as in frame #4. A lateral fibial plateau fracture was identified as in frame #2 with clinical evidence as well confirming a medial dislocation of the femur on the tibia as in frame #2 and 3. Rapid thawing of the right and left lower legs was performed in the emergency room with warm water and warm packs.

**Frame #5:** Demonstrates the patient, after thorough evaluation, being brought to surgery and the completely thawed right leg then prepped and then examined. The right lower leg was cold, edematous and pulseless well above the knee. At the same time, a puncture wound of the right groin was found with multiple lacerations of the common femoral vein. This was the result of an accidental ice ax penetration during the fall.

The Capsule of the right knee as in frame #3 was totally destroyed at the time of surgery. The popliteal artery was contused and occluded with subadventitial hemorrhage present. The injured and occluded artery was resected, and a 5 cm saphenous vein graft was taken from the left groin and sutured in place. The fasciotomy, after arterial repair, was extensive and deep, revealing that tissues in the posterior compartment of the calf and distal femur were quite swollen and edematous. A segment of the gastrocnemius was necrotic and was resected. Despite 22 hours elapse from the time of injury and since popliteal occlusion and laceration, the result was considered good with tissue loss limited to toes at the metatarsophalangeal joint level.

It is considered that by having the right leg frozen to the level of the tibial tuberosity and cool well above that, that the metabolic needs of the extremity were minimized despite the popliteal artery laceration. This allowed preservation of tissue, and along with the extensive fasciotomy, avoided compartment pressure injury and destruction of muscle, vessels and nerves.

**Frame #6:** At three weeks, the area of fascial release was covered with split thickness skin graft and the patient transferred to his native country for further care.

Fig. 17.

Patient transferred from interior Alaska three days after diving 30 feet from an oil tank with a line tied about his ankles to adjust his back, for back pain. (After viewing TV ad for traction apparatus.) Severe cold weather. Patient hung off ground, unable to undo rope for 12 hours throughout night, found at day light. Severe swelling and compartment pressure increase, with all arterial supply to limb thrombosed. Compression of supramalleolar tissues seen in above figure. All tissues below compression were nonviable. Feet frozen to level of compression.

Fig. 18. See following page.

Fig. 19.

Right foot of a fisherman with diabetes mellitus and small vessel disease, who froze his right foot; thawed aboard ship spontaneously. Eventually, because of small vessel occlusion, a metatarsal amputation was performed.



Fig. 17.



Fig. 19.



Fig. 18

Frame #1: Home in alder thicket, consisting of newspapers, cardboard, and a tattered sleeping bag. He was determined to be mentally incompetent.

Frame #2: Severe frozen feet, after 10 days of freeze-thaw-refreeze injury on at least three occasions.

Frame #3: Found to be hypothermic (27.6 degrees C.) (81.7 degrees F.), with a blood glucose of 900 mg/dl. He was warmed by peritoneal dialysis, and his frozen extremities thawed in a Hubbard tub, simultaneously.

Frame #4: Isotope technetium 99m studies demonstrated total block and failure of perfusion at the malleolar level.

Frame #5: At 72 hours the feet were pulseless, lifeless and toe gangrenous changes developing.

Frame #6: Nine days after admission, the patient developed evidence of acute bacterial sepsis, became febrile, culture demonstrating overwhelming proteus vulgaris. A guillotine low level amputation was performed.





**Fig. 20.** C-899-84 Severe Cold Exposure: Frostbite and Epiphyseal Necrosis.

**Frame #1:** This child, birthdate 6-11-81, sustained severe deep freezing injury to feet and hands, at age 2 1/2. Exposure time was greater than 4 hours, at a time of very low temperatures and heavy wind and blowing snow. When discovered by villagers, the patient apparently had extremity thawing with very hot water. The child was transferred from the northwest area of Alaska to Anchorage at one week post injury. At that time the feet were found to be edematous, pulseless, with tight constricting eschars to the level of distal tibia bilaterally. A Technetium 99m scan demonstrated a complete loss of capillary perfusion at the supramalleolar level bilaterally. Doppler examination confirmed this.

**Frame #2:** Fasciotomy was performed at the time of admission and demonstrated total blockage of the posterior tibial arterial system to mid calf.

**Frame #3:** Both hands were frozen as well, but no gross phalangeal loss occurred. However, as the x-ray indicates, taken at age 3, it is apparent that there is shortening of the distal phalanges. There is IP joint narrowing, indicating articular cartilage and epiphyseal cartilage loss of the IP joints.

**Frame #4:** Two weeks post fasciotomy, that failed to relieve the increasing necrosis, the patient had bilateral modified guillotine amputations at the level of viable and necrotic tissues, in order to preserve all available length. Skin traction was applied post-operatively. The level of amputation was supramalleolar on the left and proximal tarsal level on the right. Six months later, post-amputation, the amputation sites were revised. On the left, the below knee stump revision included excision of the fasciotomy, fixed scar. On the right, a modified Syme amputation was done.

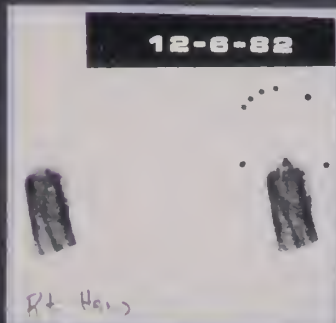
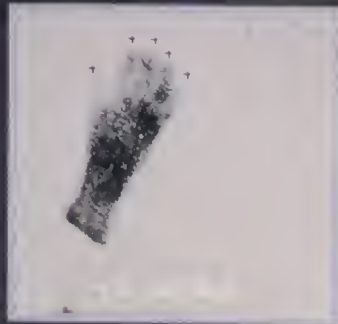
**Frame #5:** Six months after the amputation revisions, the patient was walking well, learning prosthetic limb use rapidly.

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## SUBSTANCE ABUSE-FROSTBITE-HYPOTHERMIA



**Fig. 21a.** C-802-82 #1 Substance Abuse - Frostbite - Hypothermia.

**Frame #1:** Demonstrates a 25-year-old patient found near comatose, semiconscious in a snow gully near a broken-down snowmachine. He had obvious hypothermia and frostbite to face hands and feet. The core temperature on arrival in Anchorage was 86 degrees F. (33 degrees C.), taken in the emergency room eight hours after rescue. His exposure time was said to be 12 to 18 hours, his ambient temperatures in the areas -4 degrees F. (-40 degrees C.), wind chill factor was -85 to -90 degrees F.

Wind at the time was 15 miles per hour, gusting. The patient was found with right sleeve of his coverall rolled tight over the mid-forearm and frozen to the arm, giving a total tourniquet effect for the length of time he was lying in the snow following his vehicle accident. Apparently his snow machine had tumbled off of a ridge. The patient was warmed in the field with warm blankets and then in the emergency room with a circulating warm water blanket, warm, moist inspired air and then brought to normothermia in a warm whirlpool Hubbard tank at 90 degrees F. He was, therefore, rapidly rewarmed in warm water. A toxicology screen in the emergency room demonstrated a positive test for cocaine.

**Frame #2:** Reveals a technetium isotope scan that on admission demonstrated perfusion almost to the finger tips. Compartment pressures taken on the same day varied in the hand between 37 and 52 mm Hg. The hand was warm and, in view of the technetium scan, appeared to have adequate perfusion. This evaluation is felt to have been marginal for a fasciotomy.

**Frame #3:** Three days post-admission, the right hand is thickened, edematous, totally insensitive and rapidly cooling.

**Frame #4:** The nasal frostbite appears related to the face-down position of the patient which was aggravated by 'snorting cocaine' the evening of the accident. The vasoconstriction of the nasal tissues was apparently secondary to (1) the severe contact with cold, snow and ice and (2) vasoconstriction of the nasal mucosa secondary to cocaine use allowing increased tissue cooling.

**Frame #5:** A repeat technetium scan (T99m) revealed no cellular perfusion distal to the wrist, a marked change in three days, the clinical change occurring in the past in the last six hours of that time.

**Frame #6:** Adequate response to treatment was present in feet, here demonstrated on the fourth post-injury day. Technetium 99m scan that day of the feet revealed adequate capillary perfusion.

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## VASCULAR OCCLUSION-FROSTBITE-HYPOTHERMIA-



Fig. 21b. C-802-82 #2. Vascular Occlusion - Frostbite - Hypothermia

**Frame #1:** Having demonstrated an increase in fascial compartment pressure of the hand, a fasciotomy of the mid-palm was performed demonstrating ulnar and radial artery occlusion. Clots were evacuated by small catheters, and treatment instituted with a thrombolytic enzyme, slow drip Streptokinase. Massive bleeding resulted three hours postoperatively throughout the hand and operative site.

**Frame #2:** The resolution of the nasal injury was present by the second week with the return of vascular supply. Treatment consisted primarily of the intermittent warm soaks as well as Dibenzyline therapy utilized for treatment of all frostbitten areas at 10 mg orally bid.

**Frame #3:** Because of sanguineous changes, necrosis of all tissues including blood vessels, open pack amputation above the wrist was performed with split thickness graft further added at two weeks post-amputation.

**Frame #4:** Open granulating stump was closed three weeks post-amputation.

**Frame #5:** A cross abdominal full pedicle flap was applied to the forearm amputation 2.5 months later.

**Frame #6:** The final result less than desirable. The severe loss of tissue and hand amputation on the right was considered the result of 12 to 18 hours of vascular occlusion to the hand, the depth and duration of freezing and the failure to relieve the distal vascular tree artery and vein of severe clotting.



Fig. 22.

Burn, followed by freezing, or freezing injury, after burn insult often results in tissue loss, usually from severe vessel thrombus.

### METHODS OF THAWING In Decreasing Order Of Effectiveness

#### 1. RAPID REWARMING IN WATER (90-106°F) (32.2-41.1°C)

Tub, Whirlpool bath, Crane lift platform in Hubbard Tub

#### 2. SPONTANEOUS THAWING

Room Temperature (?)  
Cabin Heat  
Thawed in foot travel or rescue Sleeping bag

#### 3. DELAYED THAWING

Ice and snow techniques  
Cold Water  
Friction massage

#### 4. THAWING BY EXCESSIVE HEAT

Camp fire heat  
Oven heat  
Engine exhaust  
Temp. (>120°F) (>48.8°C)

Fig. 23.



Fig. 24. C-144.

**Frame #1:** Froze feet barefooted in snow, temperature 0 degrees F. (-18 degrees C.), exposure 30 minutes.

**Frame #2:** Within 24 hours, huge bullae formed on plantar surface giving appearance of superficial freezing.

**Frame #3:** At patients request, because of severe formication on left sole, the bleb was ruptured. Fibrous strands formed 48 hours post freezing.

**Frame #4:** Serum content of blebs similar to normal serum exudation except for decreased protein (4.9).

**Frame #5:** Pain much increased on left after bleb rupture. No infection resulted, with continued whirlpool therapy. At five weeks both feet almost equal in pattern, with early return of sensation, the fore foot demonstrating a hypesthesia and the arch and heel evidencing hyperesthesia.

**Frame #6:** At two 1/2 months, edema still present on dependency, on ambulation. IP joints less than 50% range of motion, with volar pad loss at toes, and incomplete desquamation.

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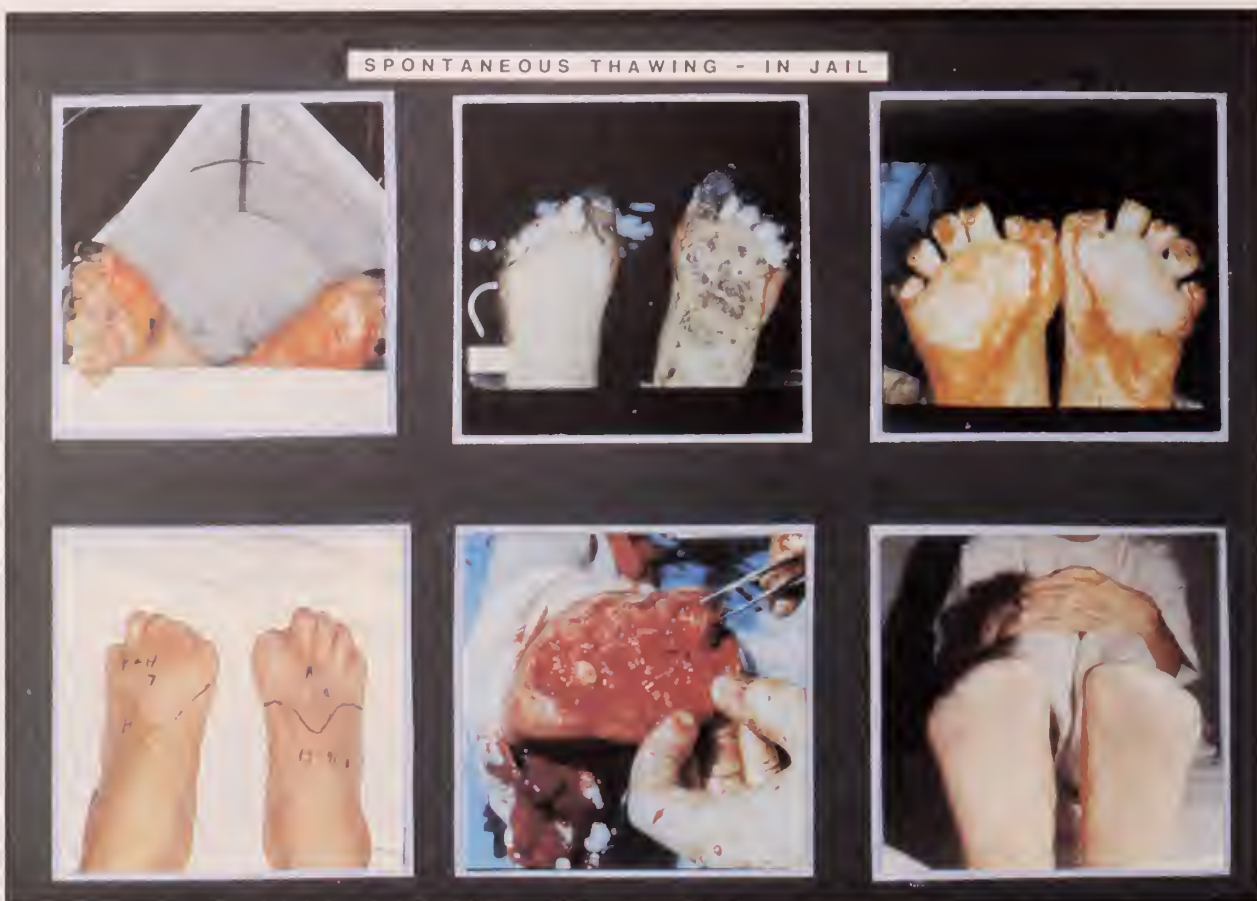


Fig. 25. C-56.

- Frame #1:** Deep frostbite, feet bilateral, after drinking episode in rural Alaska. Transferred with gangrenous toe tips by one week.  
**Frame #2:** Escaped hospital bed, irrational, and ran barefooted in snow till captured. Post-alcoholic encephalitis diagnosis.  
**Frame #3:** Necrotic tissues removed, fish mouth incision loosely closed over penrose, one suture, continued with whirlpool.  
**Frame #4:** Pain, swelling, hyperhidrosis, hypesthesia; and anesthesia residual digits.  
**Frame #5:** At revision operative procedure one and half years post injury. Avascular first metatarsal head revealed.  
**Frame #6:** Adequate weight bearing transmetatarsal amputation.

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C-43-60

# DELAYED THAWING--WITH ICE AND SNOW



Fig. 26. C-43-60. Delayed Thawing - With Ice and Snow.

**Frame #1:** The patient, a 54-year-old Alaskan Eskimo, a trapper and hunter, was on a trail in the high Arctic when a blizzard struck. His dog team ran off, leaving him stranded without food, water or shelter with the patient wearing thin cloth gloves and uninsulated rubber boots. The ambient temperature varied between -50 degrees F. (-50 degrees C.) and -20 degrees F. (-29 degrees C.). He walked for six days, his feet 'frozen solid' by the third day. He walked at least 20 miles each day with only snow as a form of fluid. His feet were frozen at least four days, and he left them in the frozen state without effort of thawing in order to maintain adequate walking and in order to survive. Upon reaching the village of Black River, his feet were immersed in snow and ice water and thawed by this delaying method over an eight hour period.

**Frame #2:** Upon arrival in Anchorage, the feet were edematous, cyanotic and cold. They were wrapped with soft dressings to (1) avoid bleb rupture and protect tissues and (2) to avoid refreezing injury.

**Frame #3:** The feet, six days post-thaw, demonstrate a sign of very poor prognosis. The blebs are dark, moderately hemorrhagic and are proximal to the MP joints. The toes and distal tissues are without blebs or blistering and are dusky, edematous, cold, and the foot is insensitive at that level. Phalangeal amputation or mid-foot amputation is generally unavoidable with this pattern. The final result might be anticipated from the date of admission, as early as 24 hours post-thaw, when blebs are large, hemorrhagic and proximal rather than pink, large and distal.

**Frame #4:** Enzyme and radioisotopes were studied as early as 1960. Here, there is an isotope evaluation procedure on the feet, using radioiodinated (I-131) Hippuran. Eventually I-125 was used in this early procedure.

**Frame #5:** By the 30th day the feet, on the frostbite regimen of twice daily whirlpool baths, demonstrate the superficial infection at the junction of the viable and gangrenous tissues. Infection is held in abeyance and controlled by whirlpool baths and aseptic care, permitting the self-demarcation of tissues, so that maximum length of foot is gained. Guillotine amputation is considered from this point on, once the tissue edema has subsided and there is no further tissue retraction.

**Frame #6:** Following revision amputation at the distal metatarsal level at three months, the patient went on to a good result and returned to his occupation of trapper and hunter.

Note: In the very early days of treatment, many patients were kept for long periods of time in hospitals. Increasing hospital expense and medical expense has caused the development of a program where after the very acute stage, the patients are sent home or to a nursing home, where post-thawing regimen including whirlpool therapy, all drugs required and digital exercises, biofeedback training are carried on at the patient's own home or in a nursing home under supervision.

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Fig. 27.

**Frame #1:** Vehicle driven off road to avoid dog team. Temperature -50 degrees F. (-45 degrees C.) ran two hours for help, in oxfords, shoes came off, and upon reaching shelter, he thawed his feet in front of a diesel generator exhaust, temperature 175 - 180 degrees F. (79.4-85 degrees C.). Seen at three weeks post injury.

**Frame #2 & 3:** At three weeks, mummification of toes and epidermal gangrenous plaques were present on soles. Multiple dorsal escharotomies performed before transfer.

**Frame #4:** Epithelialization present under black eschar of sole and heel.

**Frame #5 & 6:** Permitted to return to work and normal activity, ten months post injury.



Fig. 28. C-402.

Male, 64 years old, with a previous diagnosis of peripheral neuritis, not aware of freezing until several days had elapsed. He relates his oversight to previously demonstrated poor sensation of lower extremities. His thawing was spontaneous, in a cabin.



Fig. 29. C-402.

Plantar views of feet had demonstrated area of edema, severe bleb formation on heels, with epidermal and dermal gangrenous skin change. Rx supportive care (see x-ray changes).



Fig. 30. C-402

Lateral views of feet, three and 1/2 weeks post freezing, snowmobile trip, with injury superimposed on condition of 'alcoholic peripheral neuropathy.' 'Unaware' of cold injury till development of mummification of toes. Calcaneal views normal.



Fig. 31. C-402.

Unexpectantly, avascular changes, with collapse of calcaneal structure occurred. Asymptomatic due to neuropathy. Generally degenerative changes are not seen as early as four months post freezing..

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# PERIPHERAL NON-FREEZING COLD INJURY: IMMERSION INJURY

by: William J. Mills, Jr.<sup>(1)</sup>  
William J. Mills III<sup>(2)</sup>



## F/V JOHN N OLAF

An eighty foot shrimper from Kodiak, Alaska.

Abandoned in severe storm and icing, winds greater than 100 knots.

Crew of four left in a raft -- all lost -- area of Shelikof Straits.

January 1974.

Photo credit: Taken by Mr. Norm Holm. Courtesy Mrs. Norm Holm, Kodiak, AK

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# PERIPHERAL NON-FREEZING COLD INJURY: IMMERSION INJURY

Most of this issue is devoted to frostbite (true tissue freezing) and hypothermia (general body cooling). Less common, but equally important, and certainly as difficult to treat is immersion injury.

## GENERAL REVIEW

Immersion injury has been known by many descriptive terms such as trench foot (12,16,17), peripheral vaso-neuropathy (14), shelter foot (16), and sea boot and foxhole foot (18). The injury may be properly described as a peripheral non-freezing cold injury that results from salt or fresh water exposure, usually at low temperatures near, but not at, freezing levels (12). However, the upper level of the "low temperature" responsible for the problem has not been fixed. It has been reported that in WW II, men shipwrecked in the Gulf of Mexico, developed symptoms and signs of immersion injury syndrome, with sensory changes, after eight days exposure in water temperatures of 15.6 to 21.1 degrees C. (60-70 degrees F.) (16).

Perhaps another way to interpret this condition is to describe it as occurring after long term exposure, at temperatures lower than extremity tissue temperature, so that following continuous wet-cold (cooler) insult, heat may be transferred from the tissues to the cooler (cold) wet environment. This form of immersion event is not to be confused with tropical immersion foot or warm water immersion foot, as seen in the period of the Vietnam war. In the former condition, of tropical immersion foot, the usual exposure time was three to seven days, the water temperature producing the injury 22-32 degrees C. (70-90 degrees F.). In the latter condition, warm water immersion foot, water or wet exposure was one to three days, and the temperature producing injury 15-32 degrees C. (60-90 degrees F.) (see **fig. 1**). In contrast to other immersion effects, in colder temperatures, permanent physical changes seldom resulted, although American troops in Vietnam did often require short periods of rest or hospitalization, and removal from the wet environment. After resolution of the presenting findings (burning sensation in the feet, pain upon walking, pitting edema and erythema, transient wrinkling and hyperhydration of skin) and return to duty, these troops however, were susceptible to re-injury (1). See **Fig. 1**.

For this condition, many authors have recommended the use of silicone grease to the soles of the feet, as well



**Fig. 1.** U.S. Marine, Vietnam. Warm water immersion foot. Demonstrating pale, almost cyanotic feet with creases, wrinkling of toes and soles. Complaint of tingling and burning.

as foot care and overnight drying before re-immersion (1,18). Some troops, however, felt it risky on patrol, to sleep or rest with boots off, not knowing how rapidly they might have to move in the event of attack, consequently drying of socks and combat boots, even tropical combat boots was not obtained. This condition was seldom found in Vietnamese regular troops or the Viet Cong, who wore open sandals. It didn't appear to be a problem with Vietnamese farmers, male or female, who spent much of their life in rice paddies and a wet environment during the working day. It would appear that open footwear (i.e. sandals allowing drainage), constant activity and intermittent air drying while working, after wet exposure, plus some degree of "acclimatization" may be helpful in prevention. The use of salves, ointments and gels appear to be of some aid in avoiding macerating warm water immersion. These, however, are of minimal benefit in cold water since they are not a barrier to cold, and would have difficulty in preventing heat loss from tissues. Cold is the major agent in tissue injury, in concert with the wet environment.

## CLINICAL PATTERN

Meryman (11), Ungley and Blackwood (14) and Keatinge (9) have reviewed the striking symptoms found in immersion injury. Meryman, in describing the wet-cold problems of trench foot and immersion injury, notes that both seem to be similar syndromes. Both are associated with wet and cold, and in both, dependency and immobility are present as predisposing conditions. The authors (9,11,14) found some disagreement in the duration of exposure causing symptoms, but as in other cold injury, the insult may be a matter of time and temperature. Some symptoms are found in just a few hours, while significant tissue loss may require many days.

Immersion injury has been described as occurring in three stages (9,11,12,14). These stages are preceded by the cold, pre-rescue condition of initial insult: as "numb" extremities, swelling of feet, the feet first "red" in appearance, then changing to pale or mottled blue or black skin color and cramping of calves. After rescue and following early warming, three "stages" or clinical time zones were described.

## STAGES OF INJURY

1. The pre-hyperemic (post initial warming) stage consists of an irregular pattern of anesthesia or "stocking" anesthesia, extreme edema, "numb" extremities, and even generalized swelling of the extremities well above the malleoli. The cold, often discolored feet, still numb, appear to demonstrate pulse-less pedal arteries to palpation. Pulses in this stage may be demonstrated by Doppler examination, if not present upon manual examination. This period may last from a few hours to many days.
2. The hyperemic stage is one lasting as long as six to ten weeks. Eventually the anesthesia disappears, replaced by tingling, aching, and pain, and intermittent throbbing. Anhydrosis is present, and most spectacularly the extremity is "hot," and often painful to touch. Anhydrosis is common, sensory and motor loss is still present in irregular fashion. The redness and hyperemia persists for many weeks along with dependent rubor and elevation pallor. Intermittent cyanosis, early blister formation, and gangrene may occur.
3. The post-hyperemic stage may last for weeks and even months, and in some cases, for years. The extremities are cold sensitive, with apparent Raynaud's phenomenon or digital blanching. Edema may still occur following motionless weight

bearing and ambulation. Eventually, hyperhydrosis may develop.

Ungley (14) noted that in the post hyperemic stage, inflammation subsides, vascular tone recovers and skin temperature falls. Complete recovery must await the regeneration of the peripheral nerves involved in the cooling insult. The signs of this regeneration are the signs suggesting re-innervation of end organs and effector organs, the return of a cold-sensitive state, partial recovery of sensation often with hyperpathia and hyperhydrosis.

Late sequellae include recurrence of pain, tingling, swelling or blisters. The persistence of hyperhydrosis or a cold sensitive state, and on some occasions, circulatory deficit as well as discomfort is suggestive of vascular occlusion. Trophic ulcers, changes in bone,

Hammer toe deformity, and intrinsic muscle atrophy may be present. (In some of our Alaska series, symptoms similar to tarsal tunnel syndrome are found, with very similar findings on nerve conduction studies, this would not seem surprising, considering the changes in neurovascular bundles and the extreme persistent edema.

## PATHOPHYSIOLOGY

The pathophysiology of immersion injury was demonstrated by Blackwood and Russell in 1943 (4), Blackwood in 1944 (5), and Denny-Brown, et al (7) in 1945. Blackwell studied rat tails under temperatures and exposure conditions similar to man (3-4 degrees C.) (37.4-39.2 degrees F.) and demonstrated damage to muscle and nerve tissue after 48 hours, increasing with longer exposures. The skin and blood vessels, he thought, were more resistant to chilling. Nerve and muscle tissue had not returned to normal two months after chilling, and after that time some muscle degeneration secondary to denervation was setting in. In 1944 Blackwood (5), studying immersion victims' pathological specimens, demonstrated damage to all tissues involved. The most severe damage was found in nerve and muscle. The large arteries generally showed no marked narrowing. In some specimens, intimal and medial fibrosis was present, as well as edema. The smaller arteries demonstrated marked narrowing. Fibrous narrowing was present in small veins.

Initially, patchy acute degenerative changes were found in muscles of leg and foot, and local irreversible damage was indicated by small areas of replacement of peripheral muscle fibers by fibrous tissue. In all the cases examined, where the patient survived the exposure, some degree of nerve degeneration was present. In severe cases, this extended to knee level. Blackwood concluded that there was evidence that human findings



were similar to those of experimental animals, and eventually, unless nerve and muscles were killed at the time of initial insult, there was evidence of nerve regeneration which, however, was often slow. Denervated muscle returned to normal, providing the delay was not so long that fibrosis and irreversible degeneration had occurred.

In 1945, Denny-Brown, et al (7), in a study of low temperature effects on the sciatic nerve of the cat (at temperatures of 0.5-8 degrees C. or 32.9-46.4 degrees F.) demonstrated that the myelin and axis cylinders of mammalian peripheral nerve are selectively damaged by cold exposure, the largest being most sensitive, and the smallest the least. Damage to large motor fibers, and those conveying a sense of contact was caused by temperatures as high as 8 degrees C. (46.4 degrees F.) for intervals as short as 30 minutes. The mildest degree of damage resembles that produced by transient ischemia. The demyelinated segment of the axon cylinder degenerates and the affected myelin undergoes dissolution. Important to know was their findings demonstrating that necrosis of whole nerve bundles occurs only following freezing. For our purpose in Alaska, such information is helpful, particularly for pathology slide review, for in many cases of immersion injury in this state, land or sea, freezing injury is often superimposed on the immersion injury, and that is always suspect when severe immersion injury is presented. The certainty of freezing superimposed on immersion injury will then account for increased unexpected tissue loss (12).

## PART II

Immersion injury in our Alaska series was seen in 105 of our 1,282 cold injured patients (8%). Those cases were found in three groups:

1. Immersion injury in the waters of the North Pacific, the Gulf of Alaska, the Bering Sea, coastal waters of Southeast Alaska, the Inland Passage, Prince William Sound, Cook Inlet and Bristol Bay.
2. The second group of immersion injury victims are those found after a period of wilderness survival due to downed aircraft; the capsizing of small boat, canoe or river-raft, small boat accidents, where in all cases, the survivors were required to walk in a wet environment, usually cold, often walking in the wilderness for many days. Generally, this was done with wet shoes,

3. Immersion injury increasingly is seen in the indigent, the homeless and the mentally disturbed. These folk, often wandering in the woods or in the streets of our major cities and often intoxicated, may be exposed for days to wet, damp conditions, usually without proper clothing, without change of foot-gear, their extremities occasionally soaked with body fluids; and, of course, often during this period of wet exposure, they may be exposed to freezing temperatures.



**Fig. 2. Frame #1:** C-826-83. The patient is a 27-year-old psychotic patient lost in the coastal woods of Alaska wearing only long underwear without shoes and stockings. He apparently had been camping in a tent when driven from the tent by a bear. He was without food and water for approximately nine days. The ambient temperatures were near freezing during the day, occasionally below freezing at night. The patient developed signs of compartment pressure injuries in feet, and fasciotomy was performed on the tenth day, the day of hospital arrival and rescue. This frame demonstrates severe necrosis and liquefaction of tissues and area of fasciotomy site performed at the day of rescue.

**Frame #2:** The plantar view of feet on the 11 day with increasing necrosis.

**Frame #3:** Anterior AP view with medial malleolar needle insertion demonstrates total loss of sensation at that level.

**Frame #4:** On day 11, technetium 99 pertechnetate demonstrates loss of capillary perfusion to the level of the malleolus bilaterally. This is a more accurate assessment of blood supply than the Doppler examination previously done that gave a positive pulse at mid-arch level, possibly reflecting pulses transmitted from a higher level.

**Frame #5:** Because of overwhelming necrosis and mixed infection of both feet, a modified guillotine amputation was performed at the level of the malleolus, where eventually adequate vascular supply was found. By the sixth week, tissue granulation was present, enough to allow split thickness skin graft cover.

**Frame #6:** At both procedures (guillotine amputation and then skin grafts to obtain physiologic cover), stump traction was used postoperatively to avoid tissue retraction and loss of length. Eventually, at six weeks, revision amputation at 10 inches from the knee joint was performed with good result. His psychiatric care continues. After review of his history elsewhere, it appeared that he had wet-cold insult, immersion injury, followed by freezing.



Fig. 3. Ten days of wet cold exposure, followed by freezing, thawing and question of refreezing. Path. Specimen from C-826-83.

1. Partially degenerated internal elastic membrane.
2. Arterial Lumen
3. Disrupted, fragmented endothelial lining.
4. Early organized thrombus.
5. Tunica media, edematous disruption of elastic fibers.

## IMMERSION INJURY (WET-COLD) IN ALASKAN WATERS

It has long been known that many dangers are associated with seafaring activities, especially in northern waters. Accidents at sea are not uncommon, whether in commercial fishing, pleasure craft, naval vessels, commercial liners, freight vessels or oil tankers. The dangers include collision at sea; icing in the winter, particularly in small fishing vessels; grounding; fire at sea; vessel instability, often associated with deck overloading. Engines and electronic apparatus may fail, particularly from storm, and flooding from wave action. Incompetence and other human errors are the age-old nemesis of the seaman and a storm at sea is always a hazard. The forces of nature, especially over the world's oceans and particularly so in the northern waters, have yet to be totally conquered by even the most advanced seaman-ship and technology of man.

Over the years, Alaskans have been accustomed to reading in their newspapers, of the large number of commercial fishing boats sinking, often with loss of most or even all of the crew. The sinkings have occurred throughout the year, in the Pacific Northwest waters, the Gulf of Alaska, the sea areas south of the Aleutian chain, and the cold waters of the Bering Sea. The more inland fishing grounds of the Inland Passage, Prince William Sound and Cook Inlet, the waters of Bristol Bay have shared in the toll of sunken vessels (2,3,8).

In a series of articles on the dangers of commercial fishing in Alaska waters, the Anchorage Times (8) in 1985 revealed the Coast Guard figures regarding fishing

vessel sinkings and crew fatalities. From 1981 to 1985 an average of 72 vessels a year were lost with a total of 108 deaths. Coast Guard figures for 1991 indicated that 38 vessels were lost, 23 of which lost the entire crew. In 1992, 46 vessels were lost with 33 of the crews (15).

It has often been said (by me, also) that prior to this past decade, "a fishing vessel a week was lost in Alaska fishing waters, with most of the crews." Now, in the 1990s, it appears that we still lose almost a vessel a week, but my delving into records of vessels lost, seemed to indicate that now, in 1993, we may save many more of the crews. However, that theory may be voided, after discussion with Coast Guard employees and statisticians from NIOSH (National Institute for Occupational Safety and Health, Alaska Division of Safety Research) (10,15). It has been pointed out that new figures indicate the death toll from fishing boat sinkings may not have changed a great deal. A Coast Guard graph in support of that statement is seen in figure 4.

If there is improvement in saving crews of fishing vessels, it may be due to the following factors:

1. More vessels are currently in the Bering Sea. In an article in Pacific Fishing (13), it is said that since 1978 the fishing fleet in the Bering almost tripled from 140 to 350 vessels. Since there are almost three times the number of vessels now, and close to the same loss of life reported year after year, perhaps there has been an improvement in rescue, particularly since many of the May-day responders and rescuers are other fishing vessels. For reasons neither NIOSH or the Coast Guard can explain, not all rescues are reported.

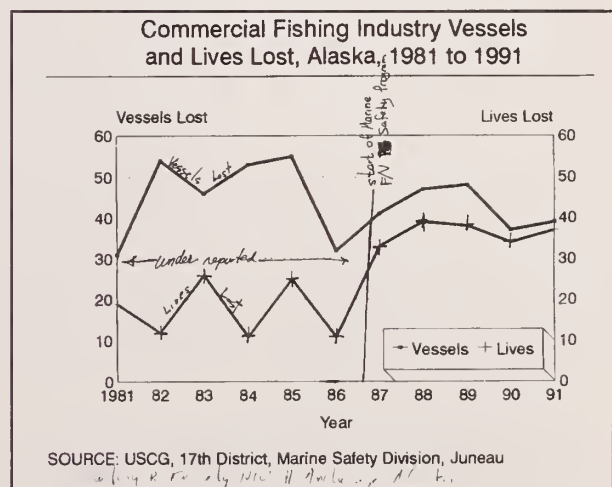


Fig. 4. U.S. Coast Guard minimal estimate of ship loses and lives lost (1981-1991) in Alaska waters.



2. More Coast Guard vessels and aircraft are available, including the C-130 and air-sea rescue helicopters.
3. More vessels have US Coast Guard approved life rafts, with double floor and canopy, containing food, water and survival gear.
4. More vessels have on board the Emergency Position Indicating Radio Beacon (EPIRB) for vessel identity and location.
5. The Coast Guard has newer, lighter, more advanced seawater pumps with greater ease of operation as compared to older pumps. They are capable of being air-dropped to sinking vessels from rescue aircraft with almost immediate utilization and operation.
6. Now more up-to-the-minute weather reports are available from the National Marine Weather Service, giving information regarding ice conditions, wind, wave action and other weather information.
7. And last, but perhaps most important for us medically, is the survival suit, a flotation unit covering all but the face, that helps in avoiding drowning and hypothermia.

## HOW DOES ALL OF THIS RELATE TO IMMERSION INJURY?

When most of the crews went down with their vessels before 1985, or perished in open rafts or while swimming the icy waters of the fishing grounds, few immersion injury victims were seen for treatment. Now that more of the crews have greater opportunity for rescue, it would appear that more wet-cold immersion problems may present for acute and long-term care. The statistics in some areas of Coast Guard research as well as from NIOSH would seem to indicate a more pessimistic attitude toward lives saved. However, in view of the outstanding rescues in recent time by the commercial fishing fleet as well as the Coast Guard, it would appear to me that when statistics are collected, there may be a greater survival rate than present statistics would indicate. The fact remains, however, that year after year in the northern waters, nearly a vessel a week still is lost.

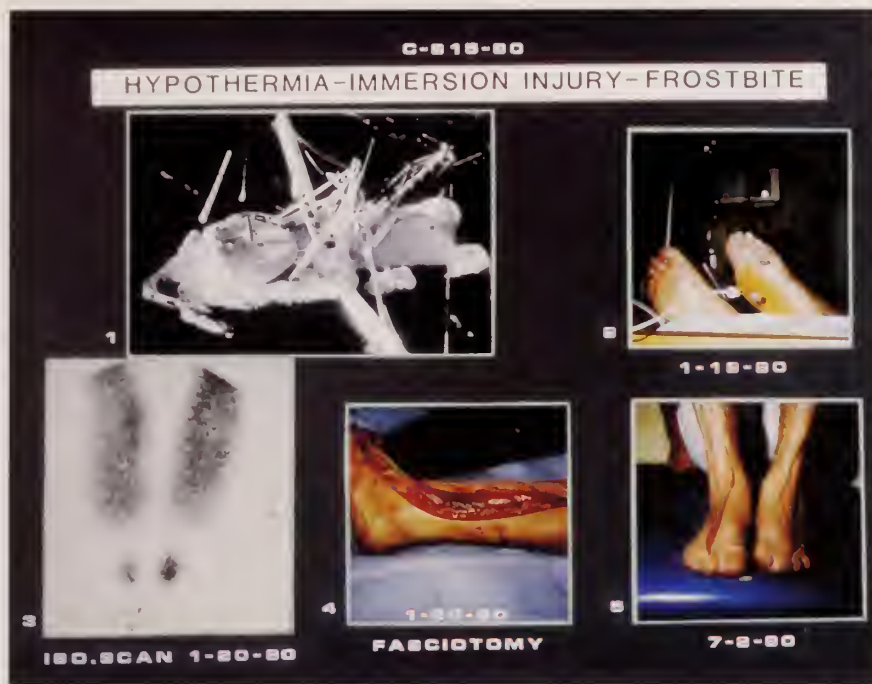
Many problems are involved in the rescue of the victim of a fishing vessel sinking. In the air or sea rescue, one must consider the transport of victims of hypothermia, immersion and frostbite injury and often other associated types of trauma including near drowning.

The physician treating the now many survivors may also be called upon to explain the causes and course of treatment for sequellae of immersion injury as well as other trauma to major insurance companies and attorneys in state and federal courts, since many of the survivors or the families of non-survivors seek legal aid and compensation. Particular interest by the representatives of both litigants, vessel owners and insurance companies has been shown in regard to etiology, pathophysiology and treatment and sequellae of immersion injury. Of no small matter is the fact that over the past few years, a fair number of most interesting cases has been referred by either insurance companies or attorneys. Their requests for examination, particularly in late stage cases, has demonstrated more than the anticipated number of sequellae in immersion cases.

What are the temperatures that fish boat survivors must face (6)? The temperatures in Shelikof Straits over the years average in January, 4-5 degrees C. (39-41 degrees F.); in February, 3-4 degrees C. (37.4-39 degrees F.); and in March, 4 degrees C. (39 degrees F.). In the cold waters of the Bering Sea near St. Paul Island, temperatures are: in January, 1 degree C. (33.8 degrees F.); in February, 1-2 degrees C. (33.8-35.6 degrees F.); and in August, 9 degrees C. (48 degrees F.). Violent winds, waves, and freezing air temperatures complicate this low sea environmental temperature for the shipwrecked fisherman. Fig. 5.

After rescue evaluation and treatment.

1. At the time of rescue, consideration must be given to the overall problem. The patient, in a survival suit or a raft, with or without a survival suit, must be considered to be hypothermic, a more major problem than immersion injury.
2. The patient must be considered to be hypovolemic and dehydrated, and possibly in some state of starvation. There may be evidence of near drowning, burns or physical trauma including fractures. It is possible that the lower extremities in the survival suit may have been lavaged with retained seawater, urine and feces. The suit itself may permit immersion changes as well as skin rash. All these conditions, in turn, need evaluation and treatment. It is helpful to determine if the tissues are frozen, prior to transport. The combined immersion and frostbite event has a much more disastrous course and prognosis.
3. When first seen by a physician, the patient is usually in the pre-hyperemic stage, or if rescue is by boat and transport is slow or delayed, the patient may be in the hyperemic stage. In the pre-hyperemic



**Fig. 5. - Hypothermia - Immersion Injury - Frostbite**

**Frame #1:** Demonstrates a fisherman, age 23, aboard a crab fishing vessel in the Bering Sea that foundered in a storm when ice accumulated on the rigging, causing the ship to 'turn-table'. The patient was in a life raft for five days, clad only in sweatshirt, pants and stockings. He was with two companions who had survival suits. The patient was without a suit. He was constantly cold and wet and was warmed by his crew companions who were in survival suits. The patient had constant wet extremities with superficial freezing of the extremities. He was hallucinating, vomiting, dehydrated with evidence of freezing of both feet at the time of helicopter rescue. Rewarming was performed in warm water at a rescue site in the Aleutian Islands, and the patient was then transferred to an Anchorage hospital. He was treated for hypothermia, by further tub warming with diagnosis of frostbite superimposed on immersion injury as well as severe dehydration and hypothermia.

**Frame #2:** The feet on admission were cold, mottled with massive edema apparent, and the patient complaining of severe pain and burning in feet. Subfascial tissue pressures indicated values throughout the feet at levels greater than 100 mm Hg.

**Frame #3:** Technetium scan the next morning after arrival demonstrates early loss of distal foot capillary perfusion.

**Frame #4:** On the same day, bilateral fasciotomy was performed, revealing only minimal bleeding at the fasciotomy site, pale tissue and evidence of early necrosis of muscle in the anterior tibial compartment. The fasciotomy permitted muscle and neurovascular structures to be relieved of occlusive pressure. Impending dusky muscle necrosis in the anterior compartment is apparent. Severe compartment pressure is further apparent as the muscle groups exploded outward after the fascia release.

**Frame #5:** Six months after injury, the patient demonstrates adequate split thickness skin graft closure, phalangeal amputation and a functional foot despite the insult of hypothermia, freezing superimposed on immersion injury, compartment pressure syndrome and dehydration and caloric deprivation. Note: Compartment pressure is common in severe cold injury. No treatment, either general or vascular or drug therapy or thawing method or sympathectomy, can be effective until the compartment pressure is relieved. Compartment necrosis greater than 37 to 40 mm Hg, especially in the range of 60 mm Hg or greater, are sufficient to occlude the capillary tree and small vessels, and this pressure must be relieved.

A further comment is that the burning, severe pain associated with immersion injury, wet-cold injury or trench foot is often relieved in the early stages by epidural blockade that is performed with an indwelling catheter for two to three days. This is further relieved by the use of the drug Dibenzylamine, an alpha blocker, given at 10 mg a day for two or three days followed by 10 mg twice daily for five to seven days.

stage there may be edema, complaints of burning and complaints of severe pressure. Cyanosis may be present. At this stage it is helpful to begin early physiotherapy, especially for joint motion of the

affected parts, and obtain a technetium 99<sup>m</sup> scan to determine the state of perfusion. If the feet are pulseless, and poor pulses are obtained by Doppler examination, consider doing immediate determination of tissue pressures on the dorsal and plantar surfaces. In some cases, swelling and edema is so overwhelming that fasciotomy is indicated. This need may be demonstrated in the early hyperemic stages.

4. In the hyperemic stage, or post-inflammatory stage, pallor may be present with cyanosis and hyperhydrosis. Physiotherapy and whirlpool twice daily should continue. At this point, the extremities may demonstrate sympathetic nerve irritation by evidence of irritability, pallor and excessive sweating. Proximal hyperesthesia is present, often with distal anesthesia. Pain often is present, particularly as edema and swelling increase. Sympatholytic drugs, as phenoxybenzamine hydrochloride (Dibenzylamine®) may be helpful, but other than supportive analgesia, the most helpful pain therapy in immersion injury has been epidural blockade, usually given in continuous mode and repeated as necessary. It is helpful to have large cradles over the feet to avoid skin contact.

5. Again in the early and even later hyperemic stage, edema and swelling may continue. You may wish to repeat the compartment pressure studies, especially if the extremity is cooler, more swollen and tense. The obvious skin ballooning and tenseness may represent sudden tissue expansion and increase in deep compartment pressure. This increase in pressure, especially in immersion injury (1,11) may cause tissue anoxemia, as well as collapse of



the small vessels and capillaries and eventually the larger vessels. This may result in severe loss of capillary perfusion with a rise in compartment pressures from 60-90 mm of mercury or more. In those cases requiring fasciotomy, the swelling and edema and pain rapidly subside (12).

6. Most patients have pain at first with weight bearing, even in the absence of blisters or swelling. Wheel chair ambulation early, followed by walker or crutch ambulation later is helpful.
7. If the patient must travel out of Alaska by aircraft, foot and leg elevation is important so that long travel with the extremities in a dependent position is avoided.

It is felt by many that immersion injury is a disease of nerves and muscle, however, in many of our patients, requiring fasciotomy or partial amputation, microscopic and tissue examination found many of the changes in blood vessels. The vascular tree is often engorged, the blood vessel lumen filled with plugs of fibrin, or by hyaline material, or even thrombus. Organized thrombi are seen in the effected areas, as well as tissues above the level of tissue damage. Blood vessel cells are swollen, often with cellular destruction of endothelium and intima and occasionally disruption of muscularis. The changes appear to be inflammatory in origin, possibly due to the action of cold on tissues, and even more likely to local hypoxia, vascular deprivation and tissue destruction. Nerves in the region reveal swelling, edema and degeneration of the axis cylinders with demyelination of the nerves and later, lipid phagocytosis.

The acute and later treatment must, therefore, include restoration of vessel patency, if isotope studies demonstrate poor perfusion, and restoration of normal circulation as a first criterion. This is so even though a period of hyperemia might indicate adequate flow. Treatment must further include a) release of tissue compartment pressure if indicated, usually by fasciotomy; b) use of drugs that promote vessel dilatation and loss of sympathetic effect, permitting drying and aiding in relieving edema pain; c) the utilization of drugs that relieve severe pain as in epidural blockade; and d) effort is directed to the prevention of infection, including the whirlpool and gentle care of the extremities. Otherwise, care is general and supportive.

## ADDENDA:

### I. Case History of Vessel Sinking and Crew Rescue (Case # C-1104-88., 1105-88, 1106-88)

History of present illness: C-1104, 1105, 1106 are rescued crewmen of a fishing vessel that sank in the Bering Sea in late winter 1988 in very stormy seas. Their vessel was electronically operated and when a particularly large wave struck the chart house, it destroyed the electronic system so that wheel and rudder became inoperable. The vessel yawed, pitched and tossed out of control, and as more water came aboard, the vessel became unstable and eventually sank, stern first.

The entire crew, wearing Imperial survival suits but cold and wet, were able to get aboard a raft. Very soon after, the raft capsized, three fishermen were able to return to the raft, but two were washed away and lost at sea. The rescued three spent 72 hours in the raft without food or water. Their survival suits contained water to calf or knee. An interesting note of their raft episode was dictated from their Anchorage hospital beds. Unbelievably, the three survivors were rescued, when in the dark of the storm, their raft bumped up against the rescue vessel.

### II. Case #1104-88, 1105-88, 1106-88, Rescue History Dictated by Survivors

"The first day we got into a life raft, our feet were wet, prior to this, and the temperature outside was in the high 30s, low 40s. The water didn't seem to be that cold because we got directly in the water. We got in the life raft and the life raft was half filled with water and like I said, our feet were already wet at the time. We got all the water out. It went into the night and each night that we were out it seemed to get a little bit colder. We were out two and one-half nights.

The first night we felt a little numbness on the feet, didn't appear to be too bad, we massaged them and kept them moving pretty good the first night and I sat in a crouched position which I think created some problems for me, cutting off circulation in my ankles.

The second day feet felt okay, just a little numb, no pain really, and the temperature outside the second day, it was a pretty nice day actually, the seas were calm and it was probably in the 40s, and the water temperature seemed to be the same, wasn't really that cold. We didn't experience any storms that day, no rain or snow, going into the night we did hit a little snow and that was probably the coldest night. I'd say it got down to maybe the mid 30s, and we were all pretty cold. Feet at that point were definitely numb but no pain really to speak of because they had gone numb. Fingers were starting

to get numb, too, index finger and the mid finger started to get real stiff and actually felt frozen.

Going into the next day, as soon as daylight came we felt warmer, fairly nice day, temperature up in the 40s again, feet by this time totally numb, massaged them off and on, didn't seem to do that much good, just felt like they were getting frozen at this point.

The third night the wind was blowing in the 70s, howling pretty good. Seas weren't that high, the wind was just blowing really steady, seas might have been only 10-15 and when we finally ran into the rescue fishing vessel, they lifted us off the raft into the boat. Immediately touching the deck everybody couldn't walk, took a couple of steps and fell down. As they took off the (survival) suits feet were totally white and blue and puffed up. Swelling was 10 to 15 times greater than normal and as the night progressed on the boat, the pain started to come as they started, I guess, to defrost; the pain got intense, more and more a burning sensation.

Got into the hospital Monday night, pain pretty intense, just a burning sensation all through the foot, the arch of the foot really burns bad, pain doesn't subside at all, it's just constant. We were given three shots of Morphine and one shot of Demerol which makes it feel better for awhile, finally knocks me out, but then as I wake up a couple of hours later the pain is so intense I have to go on Demerol the rest of the time. Today is Friday. Pain is still there, not as heavy as it was before, swelling has gone down, everything is looking pretty good."

### III. HOSPITAL COURSE

The survivors spent one week in the hospital before transfer to the lower 48 home areas. Their common signs and symptoms were markedly swollen feet, distended tissues, decreased pedal pulses with cyanosis of toes, though the feet were warm, and by the third day, hot and hyperemic. Induration of extremities was present above the malleoli. Pain and burning were present after 36 hours. Anesthesia, present on admission, soon becoming hypesthesia, with general loss of swelling after b.i.d. whirlpool and physiotherapy. Their primary problem was pain, somewhat relieved by whirlpool and analgesia, including Morphine Sulfate and Demerol. Despite the severe swelling, intercom-partmental pressures remained at levels less than 40 mm of Hg, and Doppler pulsations were present. Fasciotomy was not required. All patients refused epidural blockade, even at discharge, and after cessation of edema, all patients complained of plantar pain upon weight bearing. Isotope studies revealed adequate perfusion.

Treatment in the hospital included b.i.d. whirlpools and active foot and ankle exercises. Isotope technetium 99<sup>m</sup> evaluation, Doppler pedal pulse examination,



Fig. 6: C-1104. 12-14-88.  
Immediate post rescue. Beginning hyperemic stage. Pain, burning, hyperemia, distal cyanosis and anesthesia distally. No evidence of hammer toe deformity.



Fig 8. C-1105 12-14-88  
Early hyperemic stage. Swelling, 'redness' edema generalized. Anesthesia of toes. Plantar arch aching, pain.



Fig. 10. C-1106. 12-18-88.  
Hyperemia of foot and supra malleolar area, plantar pain upon weight bearing. Hypesthesia to anesthesia from mid foot to toes. No evidence of hammer toe deformity.



Dibenzylene®, 10 mg b.i.d. in the prehyperemic stage, and correction of hypovolemia and dehydration. All patients were given Buerger's exercises q.i.d. All required large foot cradles. All had compartment pressure measurements. At the time of initial examination, intrinsic muscle atrophy and claw toe deformity was not present. Only one patient developed a blister. Even though all temperatures were above freezing, one day their exposure included a snow storm with winds of 40-60 knots. As with all winter North Pacific and Bering Sea immersion injury, superimposed freezing is suspect. At the time of discharge, pain and edema were almost absent. Anesthesia was replaced by hypersensitivity and severe burning was no longer present. Moderate elevation seemed to help discomfort.

At the request of law firms representing the patients, examination and re-evaluation was performed in Anchorage one and 1/2 years after initial hospitalization. Subjective and objective findings are listed for each patient.



**Fig 7. C-1104 6-21-90.**  
Plantar pain, edema of feet, hyperhydrosis. Hammer toe deformity bilateral.

### CASE 1104-88 (6-21-90)

#### Subjective:

1. Discomfort worse in the winter
2. Feet and toes "burn" -- cannot touch
3. Feet feel tight after sitting long periods
4. Cannot tolerate weight of bed clothes on feet
5. Feet "feel good" in warm water
6. Sensation of "burning" in cold water
7. Mixed hypesthesia, and anesthesia, plantar arch
8. Shoe insert increasing weight on heels reduced foot ache and pain
9. Discomfort in working and attending school -  
- especially aching, foot swelling over long periods of sitting

#### Objective:

1. Remained on crutches for 2-1/2 months post injury
2. Hobbles, limps, when walking. Weight applied to external aspect of soles
3. Pedal pulses diminished
4. Marked hyperhydrosis
5. Toes cold, cyanotic
6. Toe temperatures in low 80 degrees F.
7. Marked edema of feet
8. Obvious hammer toe deformity, not present 1-1/2 years ago
9. X-rays of feet demonstrate no areas of lysis or destruction of bone
10. Technetium 99m studies negative
11. Nerve conduction studies not done



**Fig. 9. C-1105. 6-21-90.**  
Intermittent hyperemia. Hammer toe marked on right. Diminished pedal pulses.



**Fig. 11. C-1106. 6-21-90.**  
Edema mild, with diminished pedal pulses. MP joint pain. Obvious hammer toe deformity of foot.

## CASE 1105-88 (6-21-90)

### Subjective:

1. Foot appearance abnormal for six months
2. Persistent pain over dorsum of foot
3. Occasional pain in balls of feet
4. Discomfort on weight bearing: "feet ache"
5. Feet "cold" -- wears stockings to bed at night
6. In winter, feet "cold," "stiff"
7. Burning now absent
8. Feet heat sensitive, heat causes "burning"
9. Complains often of "numbness"
10. Sensation of "pins and needles" to pinwheel exam
11. Mixed hypesthesia, anesthesia of toes
12. Hyperesthesia in large toe area
13. Feet swelling in shoes, when walking
14. Unable to work

### Objective:

1. Has obvious limp
2. Intermittent hyperemia of feet
3. Diminished pedal pulses manually, but strong Doppler pulses
4. Marked hammer toe deformity not present 1-1/2 years ago
5. Red discoloration, brawny induration feet and ankles
6. EMG studies abnormal, demonstrate motor neuropathy. Nerve conduction studies demonstrate seral nerve defect, left.
7. Foot temperature measurements high 80 degrees, low 90 degrees F. One and one-half years ago, all temperatures ranged between 99-100 degrees F.

## CASE 1106-88 (6-21-90)

### Subjective:

1. Feet have changed -- "not as before", "feel tight"
2. Complains of constant pain in MP joints
3. Pain, tingling, large toes without burning
4. Feet swell after standing or ambulation
5. Can tolerate warm water
6. Cold water causes "numbness" in feet
7. Can no longer run or play sports
8. "Needle like" pain third toe, right
9. Unable to work, as foot pain requires rest every few hours
10. Complains of appearance intermittently of small nodules or "bumps"
11. Foot pain greater in left foot than right

### Objective:

1. Balance poor, walks "flat footed"
2. Superficial veins on foot not visible
3. Moderate edema and swelling, feet and ankles
4. Diminished pedal pulses
5. Strong Doppler pulses bilaterally
6. X-rays of feet suggest early lytic, periarticular lesions in phalanges, MP joint areas
7. Intrinsic small muscle contracture
8. Marked hammer toe deformity left foot
9. Nerve conduction studies demonstrate peripheral neuropathy, left greater than right
10. Technetium 99m studies demonstrate adequate perfusion feet bilaterally
11. Toe temperatures varied from 100-102 degrees F. in 1988, and had dropped to 89-91 degrees F. in 1990, 1-1/2 years later

## VI. SUMMARY OF CASE FINDINGS

1. In these patients, symptoms of injury last at least 1-1/2 years
2. Persistent pain, ache, burning remain
3. Edema and swelling, even above the malleoli are common
4. Gait is limited, limp is persistent
5. The peripheral temperatures vary, and pedal pulses appear diminished
6. Neurological signs vary from one patient to another, but in those two undergoing EMG and nerve conduction studies, there is evidence of intrinsic muscle changes and of decreased nerve conduction
7. Hypesthesia, anesthesia and hyperesthesia bordering on hyperpathia are still present
8. In one patient, early degenerative changes are beginning to appear in the MP joint areas
9. The most consistent change in these patients and many others treated, is the development of extension of the proximal phalanx at the MP joints and flexion of the middle phalanx at the PIP joints, presenting as a classical hammer toe deformity. In immersion injury, as in freezing injury, intrinsic muscle atrophy and contracture is an apparent result of low temperature.

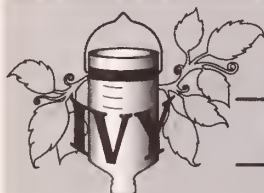


If these symptoms persist, treatment may be directed to care of hammer toe deformity, foot procedures to overcome intrinsic muscle atrophy and contracture, and correction of degenerative joint changes in MP and IP joints. Even more important is the consideration of regional (lumbar) or peripheral sympathectomy (arterial stripping of medial, lateral, plantar vessels) determined by nerve conduction and EMG studies, and measurement of vessel patency, it may be proper to perform tarsal tunnel exploration and release.

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# Psychological and Psychophysiological Factors in Prevention and Treatment of Cold Injuries

Bruno Kappes, Ph.D.<sup>(1)</sup>, William Mills, M.D.<sup>(2)</sup>, James O'Malley, M.D.<sup>(3)</sup>

*"MAN IN THE COLD IS NOT NECESSARILY A COLD MAN."* (LeBlanc)

Prolonged exposure to snow, wind, water, and/or altitude in cold temperature environments has long been recognized as the critical stressor accounting for most cold injuries. Cold can produce permanent physical and functional losses due to frostbite or fatal effects from hypothermia. When unprotected human tissue is subjected to extended or extreme cold exposure, there is an increased potential for disrupting several physiological functions and creating permanent anatomical damage as when true freezing occurs (2-5). Although extreme environmental conditions and the natural biological limitations of homothermic organisms alone are sufficient precursors to cold injuries, certain "biopsychosocial" variables increasingly account for the greatest incidence and severity of injury (6).

The curious and perplexing nature of individual differences across cold environments provides an interesting challenge for understanding psychological and physiological adaptive responses to cold. Experiences cited by pioneer polar explorers often conjures images of heroic challenges of incredible and remarkable journeys against seemingly impossible odds in the face of cold weather survival. Documented accounts of legendary military battles staged in cold environments have reported on the tragic consequences sustained by many cold casualty field troops (7). Popular entertainment films and the media have also recently rediscovered that cold survival stories sell. Contemporary films like "Alive" and "K2", and the recent news reports of a frostbitten couple with an infant stranded for days in a Nevada snow storm, all perpetuate cold fear as part of our national consciousness. Extraordinary experiences in the cold, wartime cold trauma, and controlled cold research studies will continue to increase our search for knowledge about preventing injuries as well as developing methods

to maximize human performance in cold environments.

This article reviews research on contributory risk factors, behavioral responses, and clinical issues associated with cold injury. The article briefly summarizes cold research issues for injury profiles, psychological responses, and sport applications. The review also examines applied psychophysiology and medical psychotherapy as supportive treatment methods for cold related vascular disorders. Specifically, thermal biofeedback training is highlighted as a clinical strategy in the treatment of frostbite, Raynauds and other cold induced vasospastic disorders. Most research contained herein represents several years of collaborative efforts with scientists, clinicians, students, sports enthusiasts, athletes, and patients in Alaska. It is with great respect and gratitude that we express appreciation and recognition to the many Alaskans who have made contributions to these studies.

## COLD RESEARCH METHODS

Cold research is conducted under a variety of conditions in natural, as well as artificially created cold environments, and is usually denoted by activity for sport or work. Indoor cold research methods can include a "cold pressor" test to evaluate psychophysiological responses. Experiments typically require extremities to be submerged in cold water under precise laboratory conditions. This technique challenges the limits subjects can endure experimentally induced cold pain. Cold pain tolerance is tested by having subjects or patients place their hands in ice water at 32 degrees F. (8). Other indoor studies have included whole body exposure to cold temperatures in environmentally controlled chambers or whole body immersion in cold water (9).

Outdoor field studies have carefully observed subjects immersed in cold water lakes with survival suits (10), or swimming in open seas (11) (i.e. Bering Strait five mile swim by Lynne Cox (see figure 1). Most outdoor cold research protocols maintain active monitoring of

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subject's vital internal and external physiological processes which include simultaneous behavioral observations. Such methods provide necessary protection and safeguards since medical personnel are immediately available and familiar with critical physiological limitations. These research protocols afford further safety since subjects constantly have the freedom to immediately terminate or withdraw without penalty. This tethered approach is quite different from naturalistic outdoor cold field studies where subjects freely choose to expose themselves to cold risk hazards. Subjects participating in amateur and professional sports, scientific expeditions, or outdoor professions perform with or without extensive safety considerations. Some cold researchers volunteer to serve as their own subjects. This has occurred with major international expeditions to Antarctica (12) and with local research expeditions on Mt. McKinley, Alaska (13).

The least controlled yet no less valued cold research data comes from "ex post facto" studies. These clinical studies represent data from hospitalized patients who have sustained cold injuries and retrospectively report on the events they perceive lead to injury. The inarticulate or confused patient frequently necessitates clinical inference built on estimating the probable sequence of factors responsible for injury. The data often suggest that most tragic consequences were preventable or, perhaps, could have been minimized if only precaution and good judgment had prevailed.

Research in industry and the military has long recognized that effective cold work performance requires a substantial increase in number of work hours. This results in a significantly longer time "labor factor" difference between winter versus summer working conditions. Indoor or outdoor research usually measures manual performance, dexterity, physiological body cooling, and thermal comfort during work. Comprehensive performance indicators usually include: tactile sensitivity, hand skin temperature, visual motor tasks, reaction time, mental tasks and perceptions during various cold exposures. Evidence supports increased psychological adaptation with repeated cold exposure, but does not appear to demonstrate significant or sufficient physiological acclimatization. Workers seem to tolerate cold conditions better with less pain and inconvenience after repeated experience in cold. Psychological adaptation to thermal discomfort, while desirable, may inadvertently pose greater risk of injury by further diminishing reliance on normal warning mechanisms for physiological cold stress (14).

Outdoor cold research in Finland has also studied physiological limitations over psychological adaptation by examining frostbite frequency for northern climate reindeer herders. These workers were found to exhibit greater incidence of cold injury regardless of age,

years smoking, or years of experience in cold work. The hours of snowmobile use, previous reports of "white finger" (Raynaud's predisposing symptoms), and work in the most northern regions were found to be statistically related to the greatest percentage of frostbite injury. The results also indicated prevailing wind chill index coupled with snowmobile activity was responsible for a fivefold increase in frostbite frequency. Apparently, the combined effects of wind, vibration, and cold created such a profound circulatory disturbance that susceptibility to cold injury was more pronounced. The practical realities of cold working conditions and specific behavioral practices underscore the interrelationship between activities, cold environments and injuries (15).

## COLD INJURY PROFILE

Even though most cold injured are typically described as hypothermic or frostbite "victims", true accidental injuries are rare and certain behavioral predispositions appears to seriously increase probability of injury. Research on personality profiles of frostbite patients in Alaska 1980-86 provides some consistent evidence that certain populations are particularly vulnerable to cold injury (16). The Alaskan data found the majority of severe injuries were not merely consequences of work or sport but more often related to personality disorders, alcoholism, activity, and previous injury. Significant psychological correlates to amputation or tissue loss were: Undefined Activity (.91), Psychopathology (.60), and Alcoholism (.60). Significant physiological correlates to amputation with frostbite injury included: Severity - Deep or Superficial (.73), Previous Injury (.52) and Freeze/Thaw/Refreeze Experience (.48). Results showed 80 percent of patients engaging in Undefined Activities (neither work or sport) demonstrated the greatest tissue loss, whereas only 18 percent of the patients had sustained subsequent loss due to sport or work. This suggests serious injury is more often a product of irresponsible activity.

An extensive Swedish forensic study on hypothermia also found biopsychosocial factors to be related to fatal consequences. For instance, autopsies showed drug use frequency was 67 percent for males and 78 percent for females. This study further confirms the prevalence of drug correlates to cold injury or death (17). Perhaps lifestyle choices and biopsychosocial factors do represent the best known predictors responsible for severe cold injuries. At the very least, certain predisposing factors, decision making patterns, and behavioral actions seem to facilitate the probability of experiencing a potentially permanent and undesirable result. Table 1 shows the commonly recognized activities and classic risk factors contributing to cold injury (6).

**Table 1. Cold Injury Risk Factors**

Altered Mental Status
Head injury
Alcohol & other drug abuse
Psychiatric disorders
Military personnel exposed to cold, wet climates
Outdoor sporting enthusiasts
Skiing
Mountain Climbing
Running
Snow machining
Mushing
Hunting
Boating
Skating
The homeless, and the malnourished
Elderly and newborns
Laborers and industrial workers in the cold
Oil, gas and pipeline workers
Trucking, warehousing, fishing

some of the cold related psychological and behavioral factors particular to hypothermia as a result of deteriorating consciousness (19).

**Table 2.**  
**Psychological and Behavioral Responses**  
**During Progressive Hypothermia**

Apathy, Amnesia
Confusion, Sleepiness
Delusions
Difficult Motor Coordination
Disorientation, Irritability
Hallucinations
Impaired Judgment,
Irrational Thoughts
Lethargy, Fatigue, exhaustion
Paradoxical Undressing
Semicoma
Slowed Speech
Unconsciousness
Unresponsiveness

## PSYCHOLOGICAL RESPONSES TO COLD

Psychophysiological research on predicting cold responses has shown constant prolonged cold exposure is capable of interfering with physical and psychological homeostasis. Specific pathophysiologic and metabolic consequences resulting from hypothermia have been well documented (18). For example, it is during the typical "shunting" or vasoconstrictive response that blood is directed away from extremities to vital organs in order to protect the thermal integrity of the body core. Hypothermic responses by their very nature also induce particular behavioral symptoms. Inadvertently however, these very behavioral responses increase susceptibility to frostbite and/or death. When a persistent and sufficient threshold of core temperature lowering is reached, a simultaneous and predictable sequence of psychological and behavioral responses produce the classic, insidious hypothermic pattern. The pattern follows a systematic and persistent heat loss to vital organs including the brain. A poorly regulated thermal metabolism results in progressive brain dysfunction and thereby decreases adequate or appropriate survival responses. Early detection remains critical since mental processes are essential to planning survival activities in field situations. The fundamental ability to maintain essential physical hydration requires minimum functioning mental capacity. Pain and discomfort of extremities are usual warning signals of heat loss, however numbing and shunting, distraction, and confusion can begin the cycle that disregards pending cold injury. Table 2 lists

These behavioral effects are typical consequences of successive heat loss from body core and central nervous system. Normal survival capacity is seriously compromised and an individual's prompt corrective actions become virtually impossible. Cold performance activities require a steadfast, alert state of consciousness. This includes the vigilance that accompanies a mindful respect of the prevailing deadly possibilities. When careless attitudes and behaviors prevail, cold can become a great equalizer or punisher. Again, the careless disregard of the effects of drugs and alcohol rapidly diminishes the normal capacity for timely corrective responses.

Psychologically, cold is for many people an unpleasant four-letter word. Cold is commonly associated with complaints, avoidance, discomfort and occasionally pain. Generally, people prefer warmer climates because of greater physiological and psychological compatibility, less demand for special clothing, equipment and less threat of injury. Avoidance and fear of the cold for some individuals can reach phobic proportions. This may be accompanied by panic. A learned morbid fear of the cold occurring from past negative experiences or because of a conditioned response association is called "Psychrophobia". Although the occurrence of this psychological disorder is rare, it can result in a pronounced anticipatory fear reaction to the mere thought of cold exposure. Research has focused on the development and refinement of a cold fear test for identifying people who have developed a potentially maladaptive stress reaction to cold environments. This test can serve as a



basis for selecting individuals with the highest positive cold environment compatibility (20). A pre-screening profile instrument seems appropriate and promising given the numbers of scientists, mountaineers, military, industry, and civilian populations who live, work and play in cold environments.

Other research on the mental and physical health of working populations in Arctic or subarctic cold environments have examined the increased susceptibility to sleep disturbance, headaches, depression, SAD (Seasonal Affective Disorder), cabin fever, Arctic hysteria, feelings of isolation and tendencies towards substance abuse. Specific psychiatric injuries in cold environments are unique and infrequent. The results indicate most workers display positive and healthy adjustment to cold and find comfort in extreme isolated conditions. Individuals who adapt well in such environments have sometimes been labeled "professional isolates" because they deliberately seek out remote working conditions consistent with their personal emotional needs (21). Adaptability to cold environments may be most agreeable to persons possessing a set of specific traits resembling the "Nordic personality" profile (22). Anthropological studies have found ecological, cultural, and socialization factors to be interactive correlates responsible for a personality style common to northern latitudes (23).

## **COLD INJURY IN SPORT**

The numbers of participants in cold related sports are increasing annually and consequently so too the potential for greater cold related sport injuries. Alaska has many local and international winter sporting events where opportunities for joy are mixed with an inherent risk for painful cold harm. An injury can be an unanticipated consequence of unprepared, naive sports enthusiasts who pay a heavy price for their ignorance, possibly to the extent of a final surrender to a powerful force that does not play favorites. Many have met their fate or left Alaska with permanent reminders of how unforgiving and dangerous a cold environment can be. Alaska's geographic location offers remarkable cold recreational challenges while also allowing scientists excellent cold research opportunities. Out of growing concern for injury and death in Alaska's wilderness, many dedicated health professionals have maintained active research programs aimed at prevention and treatment of cold related injuries.

Since 1980, the University of Alaska, Anchorage has been the site of the Center for High Latitude Research under the direction of William Mills, M.D., surgeon and professor. Several faculty members from different departments: Psychology, Nursing, Health Science, and internationally recognized sport physicians have supported several field research projects regarding physical

and psychological health, psychophysiology, injury prevention, and treatment protocols for cold weather sport performers. UAA research scientists have studied cold weather sport performers on the many popular cold races including: Iditarod, Iditaski, Iditabike, and Iditashoe (24-27) (see figure 2,3 &4). All these studies have examined the well debated differences between male and female cold weather performance. The data suggest that the sexes are different at different times on different races for different reasons and the debate continues. Overall, injuries have been few and minor. Risks are minimized by rules, regulations, check points, medical care and constant attention to tracking participants.

Other cold sport research projects in Alaska have focused attention on mountaineering because risk and potential injury are constant companions. Mt. McKinley, ("Denali" meaning the great one, the official Alaskan name) is North America's highest peak with an elevation of 6,194 m (20,320'). There are great numbers of climbers worldwide who successfully scale the icy, snow covered slopes at a 50 to 60 percent summit success rate. Over 1,000 climbers attempt the summit each year, 43 percent coming from foreign countries. Twenty-two rescue or recovery missions were conducted during the 1992 climbing season. The military and National Park Service spent over \$430,000 for rescues during 1992. This past climbing year saw 105 cases of acute mountain sickness (AMS) and 38 cases of frostbite. There were 11 deaths in 1992 and 75 total recorded climbing deaths from 1932 to present. Thirty-three dead bodies remain on McKinley from this 60 year period. Sport medicine research camps on McKinley have studied hypothermia, frostbite, high-altitude pulmonary edema and acute mountain sickness associated with mountaineering. Two dedicated medical research base camps were established at 7,300 ft. and 14,300 ft. (28,29)(see figure 5). Other research conducted on McKinley climbers has sought to determine the psychological and physiological factors responsible for successful summit performance and injury prevention (13). The evidence suggests there is a significantly greater mortality rate for non-guided expeditions. Since the advent of guiding, only three out of the 44 climber deaths have occurred on professionally guided expeditions. Perhaps guided ascents are safest because climbing guides are ever mindful of their liability to clients. They may choose safer routes and use more caution than non-guided teams. The most dramatic injuries, death and rescue missions have frequently included a disproportionate share of foreign born climbers. The Denali National Park Service is dedicated to education, communication and cooperative training with foreign climbing organizations to reduce the rising statistics. Injury ratios may reflect different understanding or

appreciation of specific behaviors necessary to cold survival on McKinley. Lack of insight, training or experience on McKinley's unique cold, high altitude environment may directly account for this unbalanced frequency. Despite one of the worst storms on the mountain this past year, poor critical decisions and behavioral responses seem to help explain the most tragic consequences. Although weather conditions set the stage, how one responds may make all the difference.

The international sports community has recognized the increased activity in mountain sports and supports the ever increasing field of "mountain medicine." Because over a million accidents occur in mountains every year, professionals including mountain rescue teams, trauma specialists, clinicians, emergency physicians, physiologists, sport psychologists and cold researchers have contributed to the scientific literature on prevention and treatment protocols (30).

## **APPLIED PSYCHOPHYSIOLOGY FOR COLD INJURY**

Applied psychophysiology represents a health care approach philosophy and procedure involving bio-behavioral methods in the self-regulation of physiology through learning techniques. Biofeedback training as a clinical procedure and training tool has been useful as an aid in learning to self-regulate peripheral skin temperature. Thermal biofeedback studies have examined skin temperature responses for the prevention, as well as treatment of cold injuries. Prevention studies have tested the effects of digital skin temperature training in cold environmental chambers while performing specific manual tasks (31). This award winning study reviewed the history of several indoor cold experiments and established current evidence for the effectiveness of biofeedback training for managing cold pain and increasing manual dexterity. Another Alaskan study examined the use of thermal biofeedback outdoors as an aid in temperature self-regulation for cold weather sports. Individuals were trained to consciously and voluntarily change surface skin temperature in the cold (32). Learning to improve thermal regulation in the cold was associated with less skin temperature decline and greater stability when compared to those without training or with only indoor training. All individuals when tested in the cold, regardless of training, overestimated their true temperature seven degrees higher regardless of training. Training to warm extremities appears to be a different discrimination task than learning to recognize and report actual skin temperature. The current research is also aimed at developing thermally sensitive gloves with thermistors linked to an auditory signal for early critical cold warning.

Aside from prevention, thermal biofeedback training has also been found useful in the clinical treatment of several vascular disorders. Many cardiovascular disorders including migraine, hypertension, diabetes, and Raynauds, have responded well to biobehavioral approaches (33,34)(see figure 6 & 7). Frostbite patients seem to be logical candidates for thermal biofeedback training specifically because the patient's vascular control may be irregular or dysfunctional. Biofeedback training methods require patients' active participation in the management of their disorder or injury through skill training. The use of computer technology to train patients to influence extremity blood flow has shown favorable results in the amelioration of the frequent vasospastic attacks common to cold injury, and also serves as a pain and/or anxiety management modality. Cold injuries of the extremities by nature are often accompanied by an array of vasospastic and vasoconstrictive responses depending on the severity of injury and methods used to thaw the frozen tissue.

Thermal biofeedback for frostbite injury was established in Alaska in the late 1970s and continues to be part of the Providence Hospital frostbite treatment protocol (35) (see figure 8). Physiological self-regulation via relaxation exercises and the specific feedback of thermal performance provides visual recognition of the power to influence physiology. Finger or toe temperatures are found to increase when patients are physically and emotionally relaxed. The temperature value can serve as an indirect indicator of vasodilatation. The average dorsal digital surface skin temperature at rest for most individuals is 90 degrees F., with males averaging 92 degrees F. and females 88 degrees F. However, this represents common resting temperatures while deep relaxation is found when skin temperatures reach 95 degrees F. or higher. Patients with frostbite, thermal disorders, or distressed individuals typically display extremity skin temperatures in the upper 60s or lower 70s. Vasoconstriction is regulated by the autonomic nervous system in the stress response and to neural activity during sympathetic arousal. Vasoconstriction and vasomotor tone are also mediated functions of the hormonal influence of adrenaline by the adrenal. Factors commonly found to increase vasoconstriction of blood vessels include: caffeine, nicotine, salt, sugar, stress, cold, and disease. Patients are instructed in ways to minimize further injury while participating in the behavioral management of their cardiovascular responses. This skill acquisition can be accomplished with or without medication or external heat application over successive training sessions. Many patients are able to reliably raise digital skin temperature +20 degrees F to the normal lower 90s following ten to fifteen half hour training sessions. A patient consciously assists in directing blood flow to injured areas either as an inpatient with bedside



monitor or as an outpatient by using similar portable thermal instruments at home. Increased perfusion to distal areas and decreased pain usually accompanies large increases in hand temperature. Increase in blood flow to injured areas may mean the difference between dead and live tissue.

### CASE STUDY EXAMPLE 1

A 20-year-old female patient reported having been cold exposed during substantial wind chill for several hours without gloves after locking her keys in the car. This probably resulted in superficial frostbite of her hands which were later allowed to warm indoors with forced warm air from a heater, and warm water. This patient presented with complaints of pain in both hands following most normal daily cold challenges. Hands appeared red (hyperemic), white (anemic) and blue (cyanotic), all within a few minutes of office observations. The episodic triphasic vasospasms alternated between vasodilatation, venous stasis and vasoconstriction. She reported that vasospasm was most pronounced upon touching a cold steering wheel, cold car door handle, a cold glass or when emotionally distressed. Besides the typical patriotic red, white and blue symptoms, this patient experienced painful throbbing and burning attacks almost daily in winter or summer.

This rapid discoloration pattern is the classic sign of primary Raynaud's. However in this case, it is called "Raynaud's Phenomenon" or secondary Raynaud's since it is likely secondary to cold injury. There was no reported family history or obvious evidence of preexisting Raynaud's disease. This is important in differential diagnosis since the incidence of primary Raynaud's is five times greater in females than males. Regardless of the cause, the thermal biofeedback treatment goals are the same. Patient is taught voluntary self-regulation of surface skin temperature by placing a thermistor on the dorsal surface of the middle finger with hands resting on armchair away from body trunk heat. Training can include resting hands in lap to assist radiant heat conduction to experience warming. Soon afterward however, patient is instructed to make these thermal increases without assistance. The key to success appears to be the minute, consistent and immediate performance detail provided by the feedback monitor which enables the patient to make appropriate corrections or thermal adjustments.

This patient showed typical finger-to-hand demarcation line including both thumbs. Finger temperatures ranged from 69-72 degrees F. as measured with a portable infrared monitor. Infrared thermal assessments have been used for quick evaluation, thereby reducing thermistor placement artifacts or contamination by

eliminating direct skin contact during measurement (34). Early training produced the normal 2-3 degrees F decrease or lack of significant change. The beginning sessions are often the poorest because patients have little skill in identifying or discriminating temperature values, or the ability to increase temperature at will. This skill requires a passive volition response that permits blood vessels to attain normal relaxed expansion. Following ten half hour sessions, this patient was able to increase skin temperature into mid 80s from the initial 70s at rest. Finally, following 16 sessions the patient was able to increase the temperature to above 90 degrees F. reliably in the clinic and home environment.

Home practice is an integral part of the thermal biofeedback training program so that patients can generalize their thermal response to normal cold challenges. Aside from the self-efficacy of influencing physiology by increased warmth in hands, this patient reported the added benefits of decrease in frequency of painful attacks. These painful episodes declined from 2-3 per day to one or two every two weeks or less. The patient's confidence comes from knowledge on how to increase temperature, as well as maintain less vulnerability to cold by slowing the rate of cooling.

### CASE EXAMPLE 2

A 40-year-old male patient was admitted to Providence Hospital thermal unit with frostbite injury to both feet. Patient was a veteran who had had previous multiple hospital admissions and procedures because of phlebitis involving both feet. This patient had a history of alcoholism and frequently fell asleep outdoors. Patient also slept in poorly heated trailers or on unheated porches. On this occasion, he had been intoxicated and during a blackout he likely froze his feet which were later thawed at Brother Francis Shelter. He refroze both feet later the same day. Technetium 99m studies demonstrated that, bilaterally, there was little perfusion beyond the metatarsal phalangeal joints of the feet. Despite all therapy including whirlpool, dibenzylamine, Buerger exercises, biofeedback and diet, tissue demarcated at a level consistent with initial technetium scan. Following surgery, pathological specimens revealed gangrenous changes of fibroconnective tissue with marked extensive necrosis of toes, with abscess formation.

Patient required a psychiatric consultation and provided a difficult challenge to thermal unit staff because of frequent outbursts of anger, demand for narcotics and generally uncooperative behavior. Patient also had a history of drug dependency and evidence of a personality disorder. Management of this patient involved several special needs related to the treatment of preexisting disorders.

## MEDICAL PSYCHOTHERAPY FOR COLD INJURY

Patients with traumatic thermal injuries whether they are burns, electrical, chemical or cold induced often experience considerable pain, anger, guilt, depression, and anxiety about the unknown future effects of their healing and recovery (37). Discussion of concerns, fears, and anxieties facing cold injured patients and their family systems are essential elements of good total health care management. Since the management of cold injuries frequently requires several weeks to several months in the intensive care unit, many collaborative efforts between the medical psychotherapist and health care team specialists facilitate high standards of patient care. Patients are often facing possible tissue loss, functional loss, questionable occupational future, alteration of body image, physical disability, permanent disfigurement, and several reconstructive surgeries. Total treatment process for these remarkable injuries involves health care teams appreciative of the many interrelated dynamics.

### SUMMARY

Cold injured patients in Alaska come from many sources. Although sport and work continues to provide large numbers of cold injured, most severe repeat injuries tend to reflect other biopsychosocial consequences. Certain behaviors can increase the probability of injury, however all persons living in cold climates are potential candidates. One can decrease risk by education, knowledge and intelligent behavior. Proper respect for adequate protection and hydration seem to be critical factors. Understanding the psychological, physiological and psychophysiological aspects of the cold environment performer helps refine the prevention and treatment strategies for cold injury.

Skill training with bio-behavioral methods, such as thermal biofeedback, and the value of medical psychotherapy appear to offer continued promise by facilitating physiologic recovery from injury, as well as assisting in long term rehabilitation. Both approaches increase the likelihood of a favorable healing response by soliciting active patient participation. Medical Psychotherapy for traumatic injuries can also help identify and manage cognitive emotional issues for families and patients faced with the permanent consequences of severe thermal injuries. Thermal biofeedback therapy has the potential benefit of encouraging greater self-reliance and responsibility for self-regulating overall health by integrating self-management skills regarding physiology, diet and lifestyle. Inpatient and outpatient biofeedback training offers specific influence over vascular responses

for healing, as well as providing an effective tool for pain management.

Interest in cold region habitation has continued to expand our study of human tolerance to harsh, extreme environments. Biological, psychological, sociological, and anthropological views on adaptation, habituation, acclimatization, and injury in cold environments acknowledges the role of development, learning and educated responses to cold environments (38,39). The study of health, performance, and injury prevention in extreme isolated cold environments has important strategic and scientific implications. What is learned from behavioral studies of cold survival provides an opportunity to increase our scientific knowledge and understanding. These cold research findings can assist in our future exploration of cold, underwater farming at great depths, and to far distance space travel to cold planets. The relatively new research frontier "Polar Psychology" has evolved to study how interactions with cold environments can have both positive and/or negative consequences. This research simulates the psychological factors likely to be encountered while exploring isolated cold regions of distant galaxies (40,41).

The psychological and psychophysiological correlates of cold experience appear to be a function of four interactive issues: the environment, genetic predisposition, learning or experience, and finally perception or cognition. Individual cold tolerance seems to relate heavily on sensation, perception and behavior. As Dr. Murray Hamlet, former director of cold-research division of the U.S. Army Institute of Environmental Medicine states, "cold depends on whether hell for you is a cold place or hot place." Some people, here in the North, claim that Alaska is "Heaven on Earth."

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Fig. 1. William Keatinge, M.D. and Jan Nyboer, M.D. measuring body core temperature via radio frequency telemetry during Ms. Lynne Cox's 1987 swim of the Bering Strait between USA and Russia at Little and Big Diomedes, Alaska.



Fig. 2. Kevin Kings (UAA sportsmedicine student) and Ron Christensen M.D. (Iditarod medical supervisor) measuring dehydration of Iditarod Champion Susan Butcher with a Tetrapolar Bioelectric Impedance Analyzer (TBIA), following her first victory finish in 1986.



Fig. 3. Iditaski skiers starting the 1988 330k (210miles) journey.



Fig. 4. Iditabike racers anticipating start of the 1989 trek.



Fig. 5. The 1982 Denali Medical Research Project basecamp at 7,330 ft. on the Southeast Fork of Kahiltna Glacier, Mount McKinley.



Fig. 6. Thermal biofeedback training program with frostbite patients at Providence Hospital Thermal Unit during early 1980s.



Fig. 7. Female patient with Primary Raynauds (labile vasomotor activity) with White Finger (pallor during vasospastic attack).



Fig 8. Thermal biofeedback response of a male Raynauds patients's surface skin temperature at 77.2 degrees F. (normal average is 92 degrees F. for males).



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# Letters to the Editor

## TREATMENT OF FROSTBITE

Dear Editor:

Recently, I was made aware of a practical method used by Alaskan Eskimos for treatment of frostbite. During the winter ocean harvest of seal, walrus, and polar bear for subsistence off the coast of eastern Siberia, Yup'ik Eskimo marine hunters treat frostbite of their hands by immersing them in the near-freezing water in the open gaps or channels of the frozen sea. According to Native hunters, a rewarming effect of the hands is obtained by ice water immersion. The temperature differential between the frozen hand surface of the Eskimo hunter and sea water during immersion may create a transient heat pump effect, with heat being transferred from the relatively warmer salt water to the colder exposed skin surface.

For centuries, Eskimos have been exposed to a harsh climate and adaptation may be assumed to play a role in their response to hypothermia (1). Vanggard and others have suggested that adaptation to cold stress could be the result of a local vascular change increasing the blood flow to the resting finger in which arteriovenous anastomosis play an important role in heat balance (2). Blood flow in the hands of cold-adapted Eskimos was found to be greater than that of less cold-exposed individuals (3). In addition, differences in diet composition can result in a physiologically significant change in cold tolerance (4). Moreover, environment and heredity may play a role in fluid viscosities of the joints in Eskimos (5,6).

Native hunters in Gambell, a remote village on St. Lawrence Island in the northern Bering Sea, told me they were unable to recall ill effects from their traditional way of on-the-spot immersion treatment of frostbite in the early stage. A practical guideline would be helpful for first aid of early frostbite using various methods, both traditional and otherwise. Epidemiologic investigation and clinical study may be needed to answer the question whether adverse effects could result from repeated episodes of early frostbite.

Millard Bass, DO, MPH, ScD  
1 Plaza Street, New York, NY 11217

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To the Editor:

Regarding the comments of Dr. Bass in his letter of December 1, 1992. There is little doubt that we can learn from a review of traditional Alaskan Native medical lore — not only in cold injury, but in many other areas of medicine. The application of cold water, ice, or snow was common thawing practice in native culture. This method of “thawing” is described as ‘delayed warming.’ The results vary from acceptable to very poor. I agree that an interesting study of Eskimo and Indian cold injury thawing methods could be done (by the Alaska Native Health organizations?) Animal studies need not be required. Evaluation of hundreds of Alaska Native freezing methods and the results would suffice — and provide worthwhile statistics gained by shear numbers alone over the years. You would find many variable methods, for instance, Indian tribes along the Yukon and Kuskokwim interior areas often thawed the frozen extremity in the warm gut cavity of small animals or birds (spruce hen), a form of rapid thawing.

Science, research, and modern medical dictum aside, throughout the ages the treatment that survives is “whatever works” and that which works is not necessarily the single method of choice.

William J. Mills, Jr., M.D.



# Alaska Medicine, Vol. 1, Number 1, March 1959

At the last Territorial Medical Association meeting in May 1958, the retiring president, Hugh Fate, Fairbanks, appointed a committee to investigate the feasibility of producing an Alaskan medical journal. William Whitehead, Juneau, who was soon to become the first president of the Alaska State Medical Association, was chairman of the committee.

The redoubtable William J. Mills, M.D., Anchorage, also a member of the committee, reasoned that the ultimate test was to go ahead and do it. And so they did. The first number of Volume I was edited by William O. Maddock, Anchorage, and was an instant success. It elicited praise from:

*The formidable New England Journal of Medicine which wrote both an editorial of welcome and a congratulatory letter stating, "If a state medical association with a hundred members can produce a journal like your first issue of Alaska Medicine four times a year, you will really be doing something."*

*Northwest Medicine, which had been the territorial association's official journal, wrote: "Your quality is equal to that of any of the state journals. I do not know how you did it, but the journal speaks very well for itself."*

*The Mason Clinic said: "From the attractive cover to the last article the organization and make-up is excellent. The scientific articles are unusual and interesting."*

At the time of Alaska Medicine's inception, communication was a problem in Alaska. The journal served to bridge this difficulty for the medical community. Moreover, the founders recognized the unique medical problems of the arctic and subarctic ecology, which are still pertinent. In addition, the journal would provide intercourse with the medical departments of the Armed Forces, the United States Public Health Service and other federal and state agencies.

Obviously most of the physicians in the state were involved. So many articles were submitted that it was not possible to include them all. Amazingly, sufficient advertising was obtained to more than underwrite the cost of the first issue. In fact, it was sent free to all doctors in Alaska as well as to medical school libraries and state medical societies in the United States. A second print-

ing of 1,000 copies sold for \$1.50 each.

Much had to be weighed. How often to publish? Quarterly, it was decided. What should the attitude be toward the quality and type of articles? Broad, was the answer.

The first issue ranged from Erwin S. Rabeau's "Botulism in Arctic Alaska" and A.N. Wilson and Mrs. Robert Baade's "Salmonellosis Traced to Seagulls in Ketchikan" to J.B. Deisher's "Hypnosis in General Practice" and attorney John Hughes' "State Courts as They Affect Physicians in Alaska." Other contributors were George Hale, Glenn Crawford, Rodman Wilson, Milo Fritz, Philip H. Moore, A.B. Colyar and J. Ray Langdon. Accompanying photographs of authors show surprisingly young faces, but this was 34 years ago.

Helen Whaley compiled medical news, entitled "Muktuk Morsels." Grace Cates wrote a news column for the Women's Auxiliary. Marilyn Wilkins provided cover art.

Off to a fine start, - except for the picture of "Mt. McKinley" on the cover. It was actually Mt. Brooks.

Gwynneth Gminder Wilson

(continued from page 87)

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Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalamic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it, however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

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**Contraindications:** Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

**Warning:** Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

**Adverse Reactions:** Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.<sup>1,2</sup> Also dizziness, headache, skin flushing reported when used orally.<sup>1,3</sup>

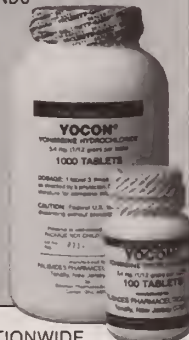
**Dosage and Administration:** Experimental dosage reported in treatment of erectile impotence.<sup>1,3,4</sup> 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to 1/2 tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.<sup>3</sup>

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**WARNINGS**

**Liver Enzymes:** HMG-CoA reductase inhibitors, like some other lipid-lowering therapies, have been associated with biochemical abnormalities of liver function. Increases of serum transaminase (ALT, AST) values to more than 3 times the upper limit of normal occurring on 2 or more (not necessarily sequential) occasions have been reported in 1.3% of patients treated with pravastatin in the U.S. over an average period of 18 months. These abnormalities were not associated with cholestasis and did not appear to be related to treatment duration. In those patients in whom these abnormalities were believed to be related to pravastatin and who were discontinued from therapy, the transaminase levels usually fell slowly to pretreatment levels. These biochemical findings are usually asymptomatic although worldwide experience indicates that anorexia, weakness, and/or abdominal pain may also be present in rare patients.

As with other lipid-lowering agents, liver function tests should be performed during therapy with pravastatin. Serum aminotransferases, including ALT (SGPT), should be monitored before treatment begins, every six weeks for the first three months, every eight weeks during the remainder of the first year, and periodically thereafter (e.g., at about six-month intervals). Special attention should be given to patients who develop increased transaminase levels. Liver function tests should be repeated to confirm an elevation and subsequently monitored at more frequent intervals. If increases in AST and ALT equal or exceed three times the upper limit of normal and persist, then therapy should be discontinued. Persistence of significant aminotransferase elevations following discontinuation of therapy may warrant consideration of liver biopsy.

Active liver disease or unexplained transaminase elevations are contraindications to the use of pravastatin (see **CONTRAINDICATIONS**). Caution should be exercised when pravastatin is administered to patients with a history of liver disease or heavy alcohol ingestion (see **CLINICAL PHARMACOLOGY: Pharmacokinetics/Metabolism**). Such patients should be closely monitored, started at the lower end of the recommended dosing range, and titrated to the desired therapeutic effect.

**Skeletal Muscle:** Rhabdomyolysis with renal dysfunction secondary to myoglobinuria has been reported with pravastatin and other drugs in this class. Uncomplicated myalgia has also been reported in pravastatin-treated patients (see **ADVERSE REACTIONS**). Myopathy, defined as muscle aching or muscle weakness in conjunction with increases in creatine phosphokinase (CPK) values to greater than 10 times the upper limit of normal was reported to be possibly due to pravastatin in only one patient in clinical trials (<0.1%). Myopathy should be considered in any patient with diffuse myalgias, muscle tenderness or weakness, and/or marked elevation of CPK. Patients should be advised to report promptly unexplained muscle pain, tenderness or weakness, particularly if accompanied by malaise or fever. **Pravastatin therapy should be discontinued if markedly elevated CPK levels occur or myopathy is diagnosed or suspected. Pravastatin therapy should also be temporarily withheld in any patient experiencing an acute or serious condition predisposing to the development of renal failure secondary to rhabdomyolysis, e.g., sepsis; hypotension; major surgery; trauma; severe metabolic, endocrine, or electrolyte disorders; or uncontrolled epilepsy.**

The risk of myopathy during treatment with lovastatin is increased if therapy with either cyclosporine, gemfibrozil, erythromycin, or niacin is administered concurrently. There is no experience with the use of pravastatin together with cyclosporine. Myopathy has not been observed in clinical trials involving small numbers of patients who were treated with pravastatin together with niacin. One trial of limited size involving combined therapy with pravastatin and gemfibrozil showed a trend toward more frequent CPK elevations and patient withdrawals due to musculoskeletal symptoms in the group receiving combined treatment as compared with the groups receiving placebo, gemfibrozil, or pravastatin monotherapy. Myopathy was not reported in this trial (see **PRECAUTIONS: Drug Interactions**). One patient developed myopathy when clofibrate was added to a previously well tolerated regimen of pravastatin; the myopathy resolved when clofibrate therapy was stopped and pravastatin treatment continued. **The use of fibrates alone may occasionally be associated with myopathy. The combined use of pravastatin and fibrates should generally be avoided.**

**PRECAUTIONS**

**General:** Pravastatin may elevate creatine phosphokinase and transaminase levels (see **ADVERSE REACTIONS**). This should be considered in the differential diagnosis of chest pain in a patient on therapy with pravastatin.

**Homozygous Familial Hypercholesterolemia.** Pravastatin has not been evaluated in patients with rare homozygous familial hypercholesterolemia. In this group of patients, it has been reported that HMG-CoA reductase inhibitors are less effective because the patients lack functional LDL receptors.

**Renal Insufficiency:** A single 20 mg oral dose of pravastatin was administered to 24 patients with varying degrees of renal impairment as determined by creatinine clearance. No effect was observed on the pharmacokinetics of pravastatin or its 3 $\alpha$ -hydroxy isomeric metabolite (SQ 31,906). A small increase was seen in mean AUC values and half-life (1/2) for the inactive enzymatic ring hydroxylation metabolite (SQ 31,945). Given this small sample size, the dosage administered, and the degree of individual variability, patients with renal impairment who are receiving pravastatin should be closely monitored.

**Information for Patients:** Patients should be advised to report promptly unexplained muscle pain, tenderness or weakness, particularly if accompanied by malaise or fever.

**Drug Interactions:** **Immunosuppressive Drugs, Gemfibrozil, Niacin (Nicotinic Acid), Erythromycin.** See **WARNINGS: Skeletal Muscle**.

**Antipyrine:** Clearance by the cytochrome P450 system was unaltered by concomitant administration of pravastatin. Since pravastatin does not appear to induce hepatic drug-metabolizing enzymes, it is not expected that any significant interaction of pravastatin with other drugs (e.g., phenytoin, quinidine) metabolized by the cytochrome P450 system will occur.

**Cholestyramine/Colestipol:** Concomitant administration resulted in an approximately 40 to 50% decrease in the mean AUC of pravastatin. However, when pravastatin was administered 1 hour before or 4 hours after cholestyramine or 1 hour before colestipol and a standard meal, there was no clinically significant decrease in bioavailability or therapeutic effect. (See **DOSEAGE AND ADMINISTRATION: Concomitant Therapy**.)

**Warfarin:** In a study involving 10 healthy male subjects given pravastatin and warfarin concomitantly for 6 days, bioavailability parameters at steady state for pravastatin (parent compound) were not altered. Pravastatin did not alter the plasma protein-binding of warfarin. Concomitant dosing did increase the AUC and C<sub>max</sub> of warfarin but did not produce any changes in its anticoagulant effect (i.e., no increase was seen in mean prothrombin time after 6 days of concomitant therapy). However, bleeding and extreme prolongation of prothrombin time has been reported with another drug in this class. Patients receiving warfarin-type anticoagulants should have their prothrombin times closely monitored when pravastatin is initiated or the dosage of pravastatin is changed.

**Cimetidine:** The AUC<sub>0-12h</sub> for pravastatin when given with cimetidine was not significantly different from the AUC for pravastatin when given alone. A significant difference was observed between the AUC's for pravastatin when given with cimetidine compared to when administered with antacid.

**Digoxin:** In a crossover trial involving 18 healthy male subjects given pravastatin and digoxin concurrently for 9 days, the bioavailability parameters of digoxin were not affected. The AUC of pravastatin tended to increase, but the overall bioavailability of pravastatin plus its metabolites SQ 31,906 and SQ 31,945 was not altered.

**Gemfibrozil:** In a crossover study in 20 healthy male volunteers given concomitant single doses of pravastatin and gemfibrozil, there was a significant decrease in urinary excretion and protein binding of pravastatin. In addition, there was a significant increase in AUC, C<sub>max</sub>, and T<sub>max</sub> for the pravastatin metabolite SQ 31,906. Combination therapy with pravastatin and gemfibrozil is generally not recommended.

In interaction studies with aspirin, antacids (1 hour prior to PRAVACHOL), cimetidine, nicotinic acid, or probucol, no statistically significant differences in bioavailability were seen when PRAVACHOL (pravastatin sodium) was administered.

**Other Drugs:** During clinical trials, no noticeable drug interactions were reported when PRAVACHOL was added to: diuretics, antihypertensives, digitalis, converting-enzyme inhibitors, calcium channel blockers, beta-blockers, or nitroglycerin.

**Endocrine Function:** HMG-CoA reductase inhibitors interfere with cholesterol synthesis and lower circulating cholesterol levels and, as such, might theoretically blunt adrenal or gonadal steroid hormone production. Results of clinical trials with pravastatin in males and post-menopausal females were inconsistent with regard to possible effects of the drug on basal steroid hormone levels. In a study of 21 males, the mean testosterone response to human chorionic gonadotropin was significantly reduced (p<0.004) after 16 weeks of treatment with 40 mg of pravastatin. However, the percentage of patients showing a  $\geq$ 50% rise in plasma testosterone after human chorionic gonadotropin stimulation did not change significantly after therapy in these patients. The effects of HMG-CoA reductase inhibitors on spermatogenesis and fertility have not been studied in adequate numbers of patients. The effects, if any, of pravastatin on the pituitary-gonadal axis in pre-menopausal females are unknown. Patients treated with pravastatin who display clinical evidence of endocrine dysfunction should be evaluated appropriately. Caution should also be exercised if an HMG-CoA reductase inhibitor or other agent used to lower cholesterol levels is administered to patients also receiving other drugs (e.g., ketoconazole, spironolactone, cimetidine) that may diminish the levels or activity of steroid hormones.

**CNS Toxicity:** CNS vascular lesions, characterized by perivascular hemorrhage and edema and mononuclear cell infiltration of perivascular spaces, were seen in dogs treated with pravastatin at a dose of 25 mg/kg/day, a dose that produced a plasma drug level about 50 times higher than the mean drug level in humans taking 40 mg/day. Similar CNS vascular lesions have been observed with several other drugs in this class.

A chemically similar drug in this class was produced optic nerve degeneration (Wallenian degeneration of retinogeniculate fibers) in clinically normal dogs in a dose-dependent fashion starting at 60 mg/kg/day, a dose that produced mean plasma drug levels about 30 times higher than the mean drug level in humans taking the highest recommended dose (as measured by total enzyme inhibitory activity). This same drug also produced vesiculocochlear Wallenian-like degeneration and retinal ganglion cell chromatolysis in dogs treated for 14 weeks at 180 mg/kg/day, a dose which resulted in a mean plasma drug level similar to that seen with the 60 mg/kg/day dose. **Carcinogenesis, Mutagenesis, Impairment of Fertility:** In a 2-year study in rats fed pravastatin at doses of 10, 30, or 100 mg/kg body weight, there was an increased incidence of hepatocellular carcinomas in males at the highest dose (p<0.01). Although rats were given up to 125 times the human dose (HD) on a mg/kg body weight basis, their serum drug levels were only 6 to 10 times higher than those measured in humans given 40 mg pravastatin as measured by AUC.

The oral administration of 10, 30, or 100 mg/kg (producing plasma drug levels approximately 0.5 to 5.0 times human drug levels at 40 mg) of pravastatin to mice for 22 months resulted in a statistically significant increase in the incidence of malignant lymphomas in treated females when all treatment groups were pooled and compared to controls (p<0.05). The incidence was not dose-related and male mice were not affected.

A chemically similar drug in this class was administered to mice for 72 weeks at 25, 100, and 400 mg/kg body weight, which resulted in mean serum drug levels approximately 3, 15, and 33 times higher than the mean human serum drug concentration (as total inhibitory activity) after a 40 mg oral dose. Liver carcinomas were significantly increased in high-dose females and mid- and high-dose males, with a maximum incidence of 90 percent in males. The incidence of adenomas of the liver was significantly increased in mid- and high-dose females. Drug treatment also significantly increased the incidence of lung adenomas in mid- and high-dose males and females. Adenomas of the eye Harderian gland (a gland of the eye of rodents) were significantly higher in high-dose mice than in controls.

No evidence of mutagenicity was observed *in vitro*, with or without rat-liver metabolic activation, in the following studies: microbial mutagen tests, using mutant strains of *Salmonella typhimurium* or *Escherichia coli*, a forward mutation assay in L5178Y TK + mouse lymphoma cells; a chromosomal aberration test in hamster cells; and a gene conversion assay using *Saccharomyces cerevisiae*. In addition, there was no evidence of mutagenicity in either a dominant lethal test in mice or a micronucleus test in mice.

In a study in rats, with daily doses up to 500 mg/kg, pravastatin did not produce any adverse effects on fertility or general reproductive performance. However, in a study with another HMG-CoA reductase inhibitor, there was decreased fertility in male rats treated for 34 weeks at 25 mg/kg body weight, although this effect was not observed in a subsequent fertility study when the same dose was administered for 11 weeks (the entire cycle of spermatogenesis, including epididymal maturation). In rats treated with this same reductase inhibitor at 180 mg/kg/day, seminiferous tubule degeneration (necrosis and loss of spermatogenic epithelium) was observed. Although not seen with pravastatin, two similar drugs in this class caused drug-related testicular atrophy, decreased spermatogenesis, spermatocytic degeneration, and giant cell formation in dogs. The clinical significance of these findings is unclear.

**Pregnancy: Pregnancy Category X:** See **CONTRAINDICATIONS**.

Safety in pregnant women has not been established. Pravastatin was not teratogenic in rats at doses up to 1000 mg/kg/day or in rabbits at doses of up to 50 mg/kg/day. These doses resulted in 20x (rabbit) or 240x (rat) the human exposure based on surface area (mg/m<sup>2</sup>). However, in studies with another HMG-CoA reductase inhibitor, skeletal malformations were observed in rats and mice. PRAVACHOL (pravastatin sodium) should be administered to women of child-bearing potential only when such patients are highly unlikely to conceive and have been informed of the potential hazards. If the woman becomes pregnant while taking PRAVACHOL (pravastatin sodium), it should be discontinued and the patient advised again as to the potential hazards to the fetus.

**Nursing Mothers:** A small amount of pravastatin is excreted in human breast milk. Because of the potential for serious adverse reactions in nursing infants, women taking PRAVACHOL should not nurse (see **CONTRAINDICATIONS**).

**Pediatric Use:** Safety and effectiveness in individuals less than 18 years old have not been established. Hence, treatment in patients less than 18 years old is not recommended at this time. (See also **PRECAUTIONS: General**.)

**ADVERSE REACTIONS**

Pravastatin is generally well tolerated; adverse reactions have usually been mild and transient. In 4-month long placebo-controlled trials, 1.7% of pravastatin-treated patients and 1.2% of placebo-treated patients were discontinued from treatment because of adverse experiences attributed to study drug therapy; this difference was not statistically significant. In long-term studies, the most common reasons for discontinuation were asymptomatic serum transaminase increases and mild, non-specific gastrointestinal complaints. During clinical trials the overall incidence of adverse events in the elderly was not different from the incidence observed in younger patients.

**Adverse Clinical Events:** All adverse clinical events (regardless of attribution) reported in more than 2% of pravastatin-treated patients in the placebo-controlled trials are identified in the table below; also shown are the percentages of patients in whom these medical events were believed to be related or possibly related to the drug:

Body System/Event	All Events %		Events Attributed to Study Drug %	
	Pravastatin (N=900)	Placebo (N=411)	Pravastatin (N=900)	Placebo (N=411)
Cardiovascular				
Cardiac Chest Pain	4.0	3.4	0.1	0.0
Dermatologic				
Rash	4.0	1.1	1.3	0.9
Gastrointestinal				
Nausea/Vomiting	7.3	7.1	2.9	3.4
Diarrhea	6.2	5.6	2.0	1.9
Abdominal Pain	5.4	6.9	2.0	3.9
Constipation	4.0	4.1	2.4	5.1
Flatulence	3.3	3.6	2.7	3.4
Heartburn	2.9	1.9	2.0	0.7
General				
Fatigue	3.8	3.4	1.9	1.0
Chest Pain	3.7	1.9	0.3	0.2
Influenza	2.4	0.7	0.0	0.0
Musculoskeletal				
Localized Pain	10.0	9.0	1.4	1.5
Myalgia	2.7	1.0	0.6	0.0
Nervous System				
Headache	6.2	3.9	1.7*	0.2
Dizziness	3.3	3.2	1.0	0.5
Renal/Genitourinary				
Urinary Abnormality	2.4	2.9	0.7	1.2
Respiratory				
Common Cold	7.0	6.3	0.0	0.0
Rhinitis	4.0	4.1	0.1	0.0
Cough	2.6	1.7	0.1	0.0

\*Statistically significantly different from placebo.

The following effects have been reported with drugs in this class:

**Skeletal:** myopathy, rhabdomyolysis.

**Neurological:** dysfunction of certain cranial nerves (including alteration of taste, impairment of extra-ocular movement, facial paresis), tremor, vertigo, memory loss, paresthesia, peripheral neuropathy, peripheral nerve palsy.

**Hypersensitivity Reactions:** An apparent hypersensitivity syndrome has been reported rarely which has included one or more of the following features: anaphylaxis, angioedema, lupus erythematosus-like syndrome, polymyalgia rheumatica, vasculitis, purpura, thrombocytopenia, leukopenia, hemolytic anemia, positive ANA, ESR increase, arthritis, arthralgia, urticaria, asthenia, photosensitivity, fever, chills, flushing, malaise, dyspnea, toxic epidermal necrolysis, erythema multiforme, including Stevens-Johnson syndrome.

**Gastrointestinal:** pancreatitis, hepatitis, including chronic active hepatitis, cholestatic jaundice, fatty change in liver, and, rarely, cirrhosis, fulminant hepatic necrosis, and hepatoma, anorexia, vomiting.

**Reproductive:** gynecostoma, loss of libido, erectile dysfunction.

**Eye:** progression of cataracts (lens opacities), ophthalmoplegia.

**Laboratory Test Abnormalities:** Increases in serum transaminase (ALT, AST) values and CPK have been observed (see **WARNINGS**).

Transient, asymptomatic eosinophilia has been reported. Eosinophil counts usually returned to normal despite continued therapy. Anemia, thrombocytopenia, and leukopenia have been reported with other HMG-CoA reductase inhibitors.

**Concomitant Therapy:** Pravastatin has been administered concurrently with cholestyramine, colestipol, nicotinic acid, probucol and gemfibrozil. Preliminary data suggest that the addition of either probucol or gemfibrozil to therapy with lovastatin or pravastatin is not associated with greater reduction in LDL-cholesterol than that achieved with lovastatin or pravastatin alone. No adverse reactions unique to the combination or in addition to those previously reported for each drug alone have been reported. Myopathy and rhabdomyolysis (with or without acute renal failure) have been reported when another HMG-CoA reductase inhibitor was used in combination with immunosuppressive drugs, gemfibrozil, erythromycin, or lipid-lowering doses of nicotinic acid. Concomitant therapy with HMG-CoA reductase inhibitors and these agents is generally not recommended. (See **WARNINGS: Skeletal Muscle** and **PRECAUTIONS: Drug Interactions**.)

**OVERDOSAGE**

There have been no reports of overdoses with pravastatin.

Should an accidental overdose occur, treat symptomatically and institute supportive measures as required. (J4-422A)



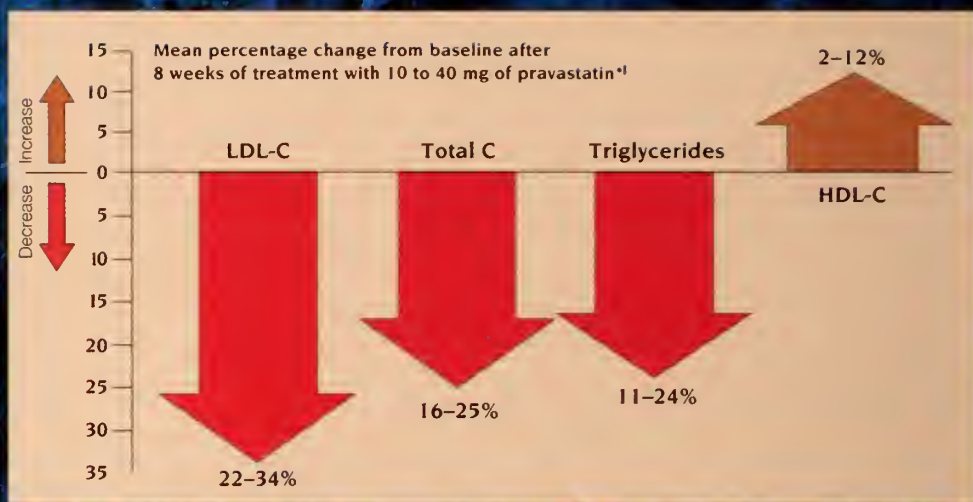


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**Reference:** 1. Jones PH, et al. Once-daily pravastatin in patients with primary hypercholesterolemia: a dose-response study. *Clin Cardiol*. 1991;14:146-151.

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# ALASKA MEDICINE

Volume 35, Number 2

April/May/June 1993



*Official Journal of:*

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*In this issue:* Alaska's Own Family Practice Residency Program by Barbara J. Doty, M.D.



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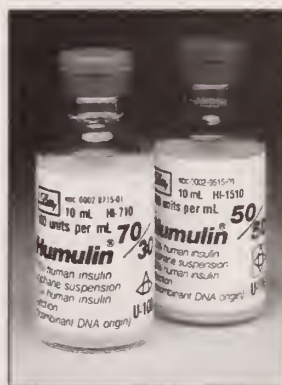
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# DEATH IN ANCHORAGE IN 1988

## and

# SUMMARY OF DEATHS 1985-1988

Rodman Wilson, M.D.<sup>(1)</sup>

### ABSTRACT

In 1988, 832 persons died in Anchorage, Alaska. Twenty-seven percent succumbed each to cardiovascular disease and cancer, 20 percent to violence including alcoholism, and the remaining 26 percent to other disorders. These proportions were about the same as in the four-year period 1985-88. Violence continued to account for more loss of life in Anchorage than nationally.

### INTRODUCTION

Eight hundred thirty-two persons died within the boundaries of the Municipality of Anchorage, Alaska in 1988. This was 23 fewer than in 1987 when estimated mid-year population of the city was 10,338 persons greater (229,117) than in 1988 (218,779) (1).

Deaths in Anchorage comprised 40.7 percent of 2,045 deaths within Alaska in 1988 (2). The population of Anchorage represented 41.2 percent of 531,000 persons estimated to be residing in Alaska in 1988.

Anchorage is the major medical center in Alaska. Many non-residents die at its two private general hospitals (Providence Hospital and Humana Hospital) or at the two federal hospitals (Alaska Native Medical Center and Elmendorf Air Force Base Hospital). Others come from smaller cities or from villages in the bush and either die violently in the streets of Anchorage or die at the homes of friends or relatives here. The number of non-residents who die in Anchorage is partially offset by Anchorage residents who die elsewhere in Alaska or in other states or countries.

### METHOD

Information was collected from death certificates and interviews with physicians as in studies of death in the three previous years (3-5). Autopsy was done in 275 (33.1 percent) instances, most commonly after violent or unexpected death. Blood alcohol values were

obtained from autopsy reports, or in a few instances from hospital clinical records.

Attending physicians or pathologists were queried in 242 (29.1 percent) cases to clarify the underlying cause of death. A final (though unofficial) cause of death was assigned to each case after careful consideration of every word on a death certificate and information gained from interviews. Examples of refined, edited, or corrected underlying causes of death are shown in Table 1.

### RESULTS

Deaths by underlying cause are listed in the Appendix.

Selected characteristics of persons dying in Anchorage in 1988 are arrayed in Table 2 together with similar data from 1985, 1986 and 1987.

Half again as many men (59.4 percent) died as women (40.6 percent) as in previous years, even though men (50.9 percent) only slightly outnumbered women (49.1 percent) in Anchorage in 1988 (1).

Median age at death was 60 years, the same as in 1987. Mean age at death, not including death before age one, was 57.9 years. In 1987, it was 58.6 years.

One-quarter (26.1 percent) of persons dying here were from out-of-town, as was the case in 1985, 1986 and 1987. Many "non-resident" infants born at Anchorage hospitals did not survive to see their home town. A newborn is conventionally assigned the place of residency of its mother.

Three-quarters (74.3 percent) of persons succumbing in Anchorage in 1988 were Caucasian and one-sixth (17.8 percent) Alaska Native. Proportions were approximately the same in previous years (Table 2).

Crude death rate (CDR) in 1988 was 3.80 deaths per 1,000 people in Anchorage. This was slightly higher than the figure 3.73 in 1987. CDR in the nation was 8.83 in 1988. "Age-adjusted" death rate (AADR), calculated by the "direct" method and using the 1940 U.S. Census as the standard population, was 7.23 in 1988 for Anchorage and 5.36 for the nation (6). Local rates, however,

<sup>(1)</sup>6234 Tanaina Drive, Anchorage, AK 99502



Table 1

**Examples of Edited Causes of Death in 1988  
Underlying Cause of Death**

Case	As recorded on certificate	As altered for this study*
017	aspiration of gastric contents	acute alcoholism
110	valvular heart disease	rheumatic heart disease
139	smoking	chronic obstructive pulmonary disease
153	chronic bronchitis	tuberculous fibrosis of lungs
222	pneumonia	fracture of hip
225	respiratory failure	lymphoma
352	cardiopulmonary arrest	intestinal obstruction
470	congenital birth defect	porencephaly
488	pneumonia of unknown origin	acquired immunodeficiency syndrome
558	thrombophlebitis	carcinoma of kidney
570	renal failure	non-A, non-B hepatitis
572	congestive heart failure	alcoholic cardiomyopathy
628	pulmonary hemorrhage	immaturity at birth
728	infiltrative liver disease	miliary tuberculosis
758	cigarette abuse	carcinoma of lung

\* see text for method

mean little because the denominator population is not that of Anchorage alone and is unknown. However, it can be noted that CDR and AARD for Anchorage in 1988 were approximately the same as CDR (3.85) and AADR (7.25) for the entire state (2).

Deaths in 1988 by broad categories of disorders are shown in Table 3, along with like data from 1985, 1986 and 1987. Deaths from "violence & adverse effects" include mortality from alcoholism. Classification of alcoholism as violence is similar to that of other studies (2) but is unlike national tabulation (6).

In 1988, deaths from cancer (225), including primary brain tumors, exceeded cardiovascular fatalities (221) for the first time since this study began in 1985, but only narrowly. Deaths from carcinoma of the lung increased to 78 in 1988. Thirty-two were among women.

Deaths from violence rose slightly to 20.3 percent in 1988 from 18.6 percent in 1987, but the increase was not statistically meaningful ( $P>.1$ ).

Table 2

**Mortality in Anchorage, Alaska, 1985-1988  
Selected Characteristics**

	1985 Number (%)	1986 Number (%)	1987 Number (%)	1988 Number (%)	Total Number (%)
Deaths	863	885	855	832	3435
Male	521 (60.4)	491 (55.5)	503 (58.8)	494 (59.4)	2009 (58.5)
Female	342 (39.6)	394 (44.5)	352 (41.2)	338 (40.6)	1426 (41.5)
Median age, years	58	59	60	60	60
Mean age, not incl age less than 1	56.7	57.9	58.5	57.9	57.8
Resident	629 (72.9)	650 (73.4)	635 (74.3)	615 (73.9)	2529 (73.6)
Non-resident	234 (27.1)	235 (26.6)	220 (25.7)	217 (26.1)	906 (26.4)
Race:					
Caucasian*	656 (76.0)	692 (78.2)	641 (75.0)	618 (74.3)	2607 (75.9)
Alaska Native	143 (16.6)	139 (15.7)	155 (18.1)	148 (17.8)	585 (17.0)
Black	36 (4.2)	31 (3.5)	34 (4.0)	39 (4.7)	140 (4.1)
Asian	18 (2.1)	13 (1.5)	22 (2.6)	23 (2.8)	76 (2.2)
Other* and unknown	10 (1.2)	10 (1.1)	3 (0.4)	4 (0.5)	27 (0.8)
Autopsy	356 (41.3)	305 (34.5)	271 (31.6)	275 (33.1)	120 (35.1)

\*including Hispanic

+including American Indian, Hawaiian, and Samoan

Twenty-nine individuals died from motor vehicle impacts occurring within Anchorage in 1988\* and 3 others died in Anchorage hospitals after crashes elsewhere. Twelve of the 29 were pedestrians and 2 others fell under wheels alighting from moving trucks. One bicyclist was struck by a car. A level of blood alcohol above 0.1 g/dL was found in 12 of 27 victims tested (mean 0.249, median 0.250, range 0.150-0.328). Seven of the 12 were pedestrians, each with a level of blood alcohol above 0.230 g/dL. The remaining 5 were all victims of single-vehicle crashes.

Thirty-seven (4.4 percent) persons died from gunfire during the year, 24 suicidally, 11 homicidally, and 2 from unintentional gunfire.

In all, there were 37 suicides and 17 homicides in 1988. Seven other individuals died directly from use of cocaine.

Forty-one (4.9 percent) individuals fell to chronic obstructive pulmonary disease (COPD). In 39 (4.7 percent) other cases COPD was listed as contributing to demise. Thus nearly 1 in 10 either died of COPD or were significantly afflicted by it.

Diabetes mellitus was deemed to be the underlying cause of death in only 11 instances but was mentioned on death certificates 41 other times. Diabetes is underreported on certificates of death both in Alaska (7) and nationally (8).

\*A thirtieth died in an Arizona hospital.

## SUMMARY OF DEATHS 1985-1988

In the four-year period 1985-88, a total of 3435\* persons died within the boundaries of Anchorage, an

average of 859 persons annually. Deaths in Anchorage comprised 41.1 percent of all deaths in Alaska, 1985-88 (2). Table 2 displays selected characteristics of persons dying and Table 3 distribution of disorders by broad categories.

No definite trends in mortality by category are noted except for a sharp fall in deaths due to violence between 1985 and subsequent years. This decrease was statistically significant ( $P<.001$ ). It came at a time when the "pipeline boom" ended and a downturn in economic activity and population occurred. At the same time, in response to the violence and high number of traffic fatalities of the mid-1980s, the budget of the Anchorage Police Department was increased and more resources devoted to apprehending drunk drivers (9).

Blood alcohol tests are available from 107 of 124 victims of lethal traffic crashes in Anchorage, 1985-88. sixty-six (62 percent) had been drinking. Blood alcohol was above 0.1 g d/L in 51 (48 percent) instances. Mean blood alcohol was 0.188 g d/L (median 0.204, range 0.017-0.381). In the nation, 49 percent of victims of motor vehicle crashes had alcohol in their blood in the years 1985-88 (10).

Thirty-seven pedestrians, including 15 Alaska Natives, were among those killed in motor vehicle encounters, 1985-88. Blood alcohol levels are available in 30 instances. Alcohol was present in 20 cases. Of 14 Natives tested, alcohol was found in 12. Experience with pedestrian fatalities mirrors that found in Alaska as a whole (11) and New Mexico (12).

There were, however, important differences in distribution of deaths by broad category relating to residency status (Table 4) and race (Table 5). One

Table 3

### Categories of Death in Anchorage 1985-1988

	1985		1986		1987		1988	
	Number	(%)	Number	(%)	Number	(%)	Number	(%)
Cardiovascular disease	242	(28.0)	269	(30.4)	265	(31.0)	221	(26.6)
Cancer	208	(24.1)	215	(24.3)	212	(24.8)	225	(27.0)
Violence & adverse effects	220	(25.5)	167*	(18.9)	159*	(18.6)	169*	(20.3)
Death before age 1	57	(6.6)	70	(7.9)	62	(7.3)	65	(7.8)
Lung disease	44	(5.1)	60	(6.8)	54	(6.3)	56	(6.7)
Infection	44	(5.1)	36	(4.1)	35	(4.1)	31	(3.7)
Neurologic disease	17	(2.0)	28	(3.2)	32	(3.7)	27	(3.2)
Gastrointestinal disease	9	(1.0)	18	(2.0)	16	(1.9)	11	(1.3)
Miscellaneous other causes	22	(2.6)	22	(2.5)	20	(2.3)	27	(3.2)
<b>Total</b>	<b>863</b>		<b>885</b>		<b>855</b>		<b>832</b>	

\*significantly different from 1985 ( $P<.02$ )



third (34.5 percent) of non-residents were Alaska Natives.

Significantly smaller proportions of persons residing elsewhere died in Anchorage from cancer and neurologic disease and a greater fraction of non-residents fell to infection, gastrointestinal disease and disorders occurring within the first year of life (Table 4).

Alaska Natives, in contrast to all other races combined, died in Anchorage less often from cardiovascular disease and cancer but more frequently from violence, fatal conditions before the age of one, infection and gastrointestinal disease (Table 5).

These variations probably have more to do with patterns of referral and city violence than to intrinsic

**Table 4**  
**Deaths by Categories of Disease Among Residents and Non-Residents**  
**Dying in Anchorage 1985-1988**

Disease Category	Total	(%)	Residents	(%)	Non-Residents	(%)	Difference*
Cardiovascular disease	997	(29.0)	717	(28.4)	280	(30.9)	ns
Cancer	860	(25.0)	666	(26.3)	194	(21.4)	P<.004
Violence and adverse effects	715	(20.8)	534	(21.1)	181	(20.0)	ns
Death before age 1	254	( 7.4)	164	( 6.5)	90	( 9.9)	P<.001
Lung Disease	214	( 6.2)	166	( 6.6)	48	(5.3)	ns
Infection	146	( 4.3)	87	( 3.4)	59	( 6.5)	P<.0001
Neurologic disease	104	( 3.0)	89	( 3.5)	15	( 1.7)	P<.005
Gastrointestinal disease	54	( 1.6)	32	( 1.3)	22	( 2.4)	P<.02
Other	91	( 2.6)	74	( 2.9)	17	( 1.9)	ns
<b>Total</b>	<b>3435</b>		<b>2529</b>		<b>906</b>		

\*chi-square: not significant P>.05

**Table 5**  
**Deaths by Category Among Native Alaskans and Non-Natives**  
**Dying in Anchorage 1985-1988**

Disease Category	Total	(%)	Non-Native	(%)	Native	(%)	Difference*
Cardiovascular disease	997	(29.0)	879	(30.8)	118	(20.2)	P<.001
Cancer	860	(25.0)	746	(26.2)	114	(19.5)	P<.001
Violence and adverse effects	715	(20.8)	565	(19.8)	150	(25.6)	P<.002
Death before age 1	254	( 7.4)	189	( 6.6)	65	(11.1)	P<.001
Lung disease	214	( 6.2)	175	( 6.1)	39	( 6.7)	ns
Infection	146	( 4.3)	110	( 3.9)	36	( 6.2)	P<.02
Neurologic disease	104	( 3.0)	79	( 2.8)	25	( 4.3)	ns
Gastrointestinal disease	54	( 1.6)	36	( 1.3)	18	( 3.1)	P<.002
Other	91	( 2.6)	71	( 2.5)	20	( 3.4)	ns
<b>Total</b>	<b>3435</b>		<b>2850</b>		<b>585</b>		

\*chi-square: not significant P>.05

biologic differences between residents and non-residents and Alaska Natives and other races.

One hundred sixty-six (18.3 percent) of 906 non-residents were from out-of-state, including 7 from other nations. Two fifths were from California, Washington and Oregon. Many more were male (62.7 percent) than female (37.2 percent). Median age was 67 years. Mean age, excluding two "non-resident" infants dying within a few hours after birth here, was 64.4 years. Both numbers are considerably higher than overall experience (Table 2).

Out-of-state residents dying in Anchorage were overwhelmingly Caucasian (89.8 percent). Two thirds (65.2 per cent) had been in Alaska for one month or less when they perished. Half (51.2 percent) of out-of-state victims died in June, July and August, the season of highest tourist, construction and commercial fishing activity. Summer concentration of deaths, short stay, race and advanced age of out-of-state victims suggest that many, if not most, were tourists.

Half (50.6 percent) of out-of-state residents dying here succumbed to cardiovascular conditions. A quarter (24.7 percent) died violently, including 10 individuals in air or traffic mishaps, 9 by gunfire, 7 from on-job injuries, 4 by drowning, 4 from alcoholism and 7 from various other misadventures including an instance in which a tourist was crushed by a moving block of ice on a glacier. An eighth (12.0 percent) died from malignant neoplasms and an eighth (12.7 percent) from a variety of other causes.

Three hundred fifty-five residents of Anchorage are known to have died away from their home city in the period 1985-88. I shall report them separately.

The five leading causes of death in the nation in the years 1985-88, namely, diseases of the heart, malignant neoplasms, cerebrovascular diseases, accidents, and chronic obstructive pulmonary diseases were similarly the leading causes of death in Anchorage during the quadrennium but ranking except for fifth place is different (Table 6). Thereafter, ranking among the next 15 common causes of death varies considerably. Sudden infant death syndrome and acute alcoholism were not among the 20 leading causes of mortality in the nation (6); death from

nephritis and vascular insufficiency of the intestine were not among the 20 commonest mortal disorders in Anchorage.

\*Duplicate death certificates in two instances in 1986 (4) were eliminated, leaving 885 deaths for that year.

## COMMENT

Table 7 lists selected common causes of demise in Anchorage by year from the period 1985-88, comprising two thirds of all deaths. Incidence of most of these conditions was remarkably static, but mortality from carcinoma of the lung appears to have been rising ( $P<.1$ ) and motor vehicle deaths fell sharply after 1985 as discussed above

It is important to note that almost all the disorders listed in Table 7 are preventable by alteration of

**Table 6**  
**Twenty Leading Causes of Death in United States and Anchorage 1985-1988**

Cause of Death	Rank	
	United States	Anchorage
Diseases of heart	1	2
Malignant neoplasms	2	1
Cerebrovascular diseases	3	4
Accidents and adverse effects	4	3 *
Chronic obstructive pulmonary diseases and allied disorders	5	5
Pneumonia and influenza	6	10
Diabetes mellitus	7	17
Suicide	8	6
Chronic liver diseases	9	8
Atherosclerosis	10	14
Nephritis, nephrotic syndrome and nephrosis	11	
Homicide and legal intervention	12	9
Septicemia	13	20
Certain conditions originating in the perinatal period	14	7
Aneurysm of aorta	15	15
Senile dementia and Alzheimer's disease	16	13
Congenital anomalies	17	11
Pulmonary embolism	18	15
Acquired immunodeficiency syndrome	19	19
Vascular insufficiency of intestine	20	
Sudden infant death syndrome		12
Acute alcoholism		18

\*alcoholism not included for this ranking only



**Table 7**  
**Selected Causes of Death in Anchorage**  
**1985-1988**

Cause of Death	1985	1986	1987	1988	Total
Coronary heart disease	147	162	153	129	592
Cerebral thrombosis	29	48	35	29	141
Aneurysm of aorta	5	6	10	6	28
Pulmonary embolism	9	7	10	3	29
Carcinoma of lung	60	66	71	78	275
Carcinoma of breast	22	25	11	19	77
Carcinoma of colon and rectum	20	15	18	21	74
Chronic obstructive pulmonary disease	37	44	43	41	165
Asthma	3	4	4	3	14
Immaturity	18	31	21	29	99
Sudden infant death syndrome	15	10	16	11	52
Acquired immunodeficiency syndrome	3	7	7	7	24
Alcoholism	46	40	40	47	173
Suicide	40	38	30	37	145
Homicide	18	18	20	17	73
Gunshot, total	39	39	30	37	145
Motor vehicle collision	58	36	27	33	154*
vehicle-pedestrian <sup>+</sup>	9	8	8	12	37

\*Fatal event occurred within Anchorage in 124 instances.

+All events occurred within Anchorage.

confirm clinical diagnosis with autopsy and to make certain that changes in underlying cause of death found at post-mortem examination amend the death certificate. This is rarely done (22).

Another way to improve accuracy is to query clinicians and pathologists about recorded causes of death (23,24). This was the method I used in these studies. To the extent that it was successful, the accuracy and usefulness of information about death in Anchorage during the years 1985-88 has been heightened. At a minimum, I have delineated mortality in Anchorage during the mid-to-late 1980s. This may interest physicians, biologists, sociologists and others in the future.

\*cases of alcoholic pancreatitis not available nationally

## CONCLUSION

In 1988, 832 persons died in Anchorage, Alaska. In the four-year period 1985-88, 3435 persons succumbed here. This was 41.1 per cent of all deaths occurring in Alaska during these years. Overall, one quarter died from cardiovascular disease, one quarter from malignant neoplasms, one fifth from violence including alcoholism and the rest from other conditions. Most disorders causing death in Anchorage could either have been prevented or detected early enough to have been forestalled.

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personal habits or are detectable at a stage when treatment is usually effective.

If all deaths from 1985-88 due to violence are combined, i.e., accidents, suicide, homicide and alcoholism, the fraction of deaths due to violence, unadjusted for age, was two and one-half times as high in Anchorage (20.8 percent) as in the nation (7.9 percent) (6\*,13), but not so high as the Baffin region of Canada (26.0 percent) (14) or in Greenland (28.8 percent) (15). Adjusted for age, the death rate from violence (0.87/1,000) in Anchorage was still 38 percent higher than in the nation (0.63/1,000).

Certificates of death are notoriously inaccurate (16-20). Clinical diagnosis at death does not match anatomical findings at autopsy in as many as 40 percent of cases. Cardiovascular diseases are overdiagnosed and pulmonary and gastrointestinal disorders underdiagnosed as causes of death.

Nonetheless, death certificates remain a rich repository of information vital for clinical and public health purposes (21). This is why it is desirable to

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## APPENDIX

### Underlying Causes of Death of 832 Persons Dying in Anchorage in 1988 (code numbers are from ICD-9-CM, 3rd ed., 1989)

CARDIOVASCULAR DISEASE		221 (26.6%)
Heart Disease*		154
coronary atherosclerosis (410,414)		129
acute myocardial infarction (410)	61	
cardiomyopathy, idiopathic (425.4)		6
rheumatic (393-398)		4
mitral valve incompetence, not otherwise specified (424.0)		2
ruptured mitral chordae tendineae, myxomatous (429.5)		2
aortic valvular stenosis, idiopathic (424.1)		2
hypertrophic obstructive cardiomyopathy (425.1)		2
unspecified heart disease (429.9)		2
hypertensive (402)		1
Eisenmenger's defect (745.4)		1
congenital heart disease, unspecified (746.9)		1
Wolff-Parkinson-White syndrome (426.7)		1
conduction disorder, unspecified (427.9)		1

\*excluding alcoholic cardiomyopathy, infections of the heart, and death from heart disease before age 1



**Vascular Disease****67**

cerebral thrombosis (434.0)	29
cerebral hemorrhage	15
intraparenchymal (431)	8
berry aneurysm (430)	6
arteriovenous aneurysm (747.81)	1
cerebral embolism (430.1)	1
aortic aneurysm	7
abdominal, ruptured (441.3)	5
thoracic, ruptured (441.1)	1
dissecting (441.0)	1
atherosclerosis, generalized (440.9)	6
pulmonary embolism (415.1)	3
atherosclerosis of vessels of extremities (440.2)	2
mesenteric infarction (557.0)	2
aortoiliac obstruction (444.0)	1
aneurysm of iliac artery, ruptured (442.2)	1

**CANCER****225 (27.0%)**

Lung (162)	78
Colorectal (153,154)	21
Breast (174)	19
Pancreas (157)	13
Lymphoma (200-202)	11
Disseminated, primary site unspecified (199.0)	11
Prostate (185)	10
Brain, primary (191)	8
Oral and nasopharyngeal (146-149)	7
Bladder (188)	6
Stomach (151)	5
Melanoma (172)	5
Ovary (183)	4
Kidney (189)	3
Myeloid leukemia (205)	3
Myeloma (203)	2
Tongue (141)	2
Esophagus (150)	2
Cervix (180)	2
Leiomyosarcoma, intraabdominal (158.8)	2
Liver, primary (155.0)	1
Gallbladder (156.0)	1
Bile ducts (235.3)	1
Ampulla of Vater (156.2)	1
Ureter (189.2)	1
Endometrium (182)	1
Anus (154.3)	1
Lymphoid Leukemia (204)	1
Myeloproliferative disorder (238.7)	1
Plasma cell disease, macroglobulinemia (203.8)	1
Angioimmunoblastic lymphadenopathy (202.9)	1

**VIOLENCE AND ADVERSE EFFECTS****169 (20.3%)****Alcoholism\*****47**

alcoholic liver disease (571.0-571.3)	24
acute alcoholism (303.0)	7

alcoholic cardiomyopathy (425.5)	5
pancreatitis, alcoholic (577.0,577.1)	5
hypothermia, drunk (E901.0)	3
delirium tremens (291.0)	1
chronic alcoholism (303.9)	1
head injury from jump from boat, drunk (E835)	1

\*not including fetal alcohol syndrome

<b>Vehicular Impacts</b>	<b>37</b>
automobile, truck (E811,E812,E815,E816)	15
auto-pedestrian (E814)	12
aircraft (E841)	3
motorcycle (E812.2)	2
fall under truck (E818)	2
bicycle-auto (E813)	1
3-wheeler hit by aircraft (E844)	1
sled hit tree (E917)	1
<b>Suicide</b>	<b>37</b>
gunshot (E955)	24
motor vehicle exhaust (E952.0)	5
hanging (E953.0)	4
drug (E950.1,E950.3)	4
<b>Homicide</b>	<b>17</b>
gunshot (E965)	11
stabbing, hatcheting (E966)	4
bludgeoning (E968.2)	1
fist (E960)	1
<b>Poisoning, Unintentional</b>	<b>10</b>
cocaine (E855.2)	7
lidocaine (E855.2)	1
methanol (E860.2)	1
multiple drugs (E858.8)	1
<b>Fire (E980)</b>	<b>7</b>
<b>Fall</b>	<b>5</b>
from stationary vehicle on job (E844.9)	1
from being hit by cable on job (919.6)	1
knocked from ladder on job (E917)	1
at home, fracture of hip (E888)	1
on road, contusion of brain (E888)	1
<b>Other Head Injury</b>	<b>3</b>
hit by tray of fish on job (E919)	1
hit by ice falling from roof (E916)	1
unexplained (E917)	1
<b>Gunshot, Unintentional (E922)</b>	<b>2</b>
<b>Miscellaneous</b>	<b>4</b>
Drowning (E910)	1
Hanging by curtain cord, unintentional (E913.8)	1
Drug overdose, multiple, undetermined whether suicidal or unintentional (E980)	1
Hypothermia, disoriented after ingestion of several drugs (E901.0)	1

<b>DEATH BEFORE AGE 1*</b>	<b>65 (7.8%)</b>
Immaturity at birth (765,765.0,765.1)	29
Sudden infant death syndrome (798.0)	11



Congenital anomalies	7
hydranencephaly (740.0)	1
hydrocephaly (742.3)	1
tetralogy of Fallot (745.2)	1
mitral and subaortic stenosis (746.3,746.81)	1
esophageal stricture (750.3)	1
diaphragmatic hernia (756.6)	1
cystic kidney disease (753.1)	1
Respiratory distress syndrome (769)	5
Viral pneumonia (480.1,480.9)	4
Viral myocarditis (422)	1
Bacterial endocarditis (421.0)	1
Meconium aspiration (770.1)	1
Pulmonary immaturity (777.4)	1
Bronchopulmonary dysplasia (770.7)	1
Hydrops fetalis (778.0)	1
Escherichia coli meningitis (320.8)	1
Necrotizing enterocolitis (777.5)	1
Fetal alcohol syndrome (760.71)	1

**\*not including 1 death each from brain tumor, homicide and fire**

<b>LUNG DISEASE+</b>	<b>56 (6.7%)</b>
Chronic obstructive pulmonary disease (496)#	41
Restrictive lung disease secondary to tuberculosis (011.4)	4
Asthma (493)	3
Bronchiectasis (494)	2
Bronchopulmonary dysplasia (770.7)	2
Rheumatoid lung disease (714.81)	1
Idiopathic fibrosing alveolitis (516.3)	1
Eosinophilic lung disease (518.3)	1
Churg-Strauss disorder (799.8)	1

**+ excluding pneumonia and pulmonary disorders causing death before age 1**  
**# COPD was listed on 39 other certificates as a significant associated condition.**

<b>INFECTION°</b>	<b>31 (3.7%)</b>
Acquired immunodeficiency syndrome (042)	7
Pneumonia (481,484,486)	7
Viral hepatitis, non-A, non-B (070.4,070.5)	4
Septicemia (038)	4
Bacterial endocarditis (421.0)	3
Viral myocarditis (422)	2
Viral encephalitis (049.9)	1
Viral hepatitis A (070.0)	1
Miliary tuberculosis (018)	1
Botulism (005.1)	1

**°not including death from infection before age 1**

<b>NEUROLOGIC DISEASE*</b>	<b>27 (3.2%)</b>
Senile dementia and Alzheimer's disease (290.0,330.1)	9
Multi-infarct dementia (290.4)	4
Epilepsy (345)	4

Parkinson's disease (332)	2
Amyotrophic lateral sclerosis (335.20)	2
Porencephaly, congenital (742.40)	2
Cerebral palsy (343)	1
Psychomotor retardation (381.2)	1
Pseudobulbar palsy (335.23)	1
Sleep apnea (780.53)	1

\* excluding cerebral thrombosis, cerebral hemorrhage, brain tumor and other neurologic death before age 1

<b>GASTROINTESTINAL DISORDERS</b>	<b>11 (1.3%)</b>
Pancreatitis, hemorrhagic (577.0)	3
Intestinal obstruction due to adhesions (560.81)	3
Esophagitis (530.1)	1
Volvulus of stomach with perforation (537.8)	1
Gastric ulcer, bleeding (531.0)	1
Duodenal ulcer, bleeding (532.0)	1
Cholangitis, acute (576.1)	1

<b>MISCELLANEOUS DISORDERS</b>	<b>27 (3.2%)</b>
Diabetic end-stage renal disease* (250.4)	9
Senility (797)	3
End-stage renal disease, non-diabetic (583.2,585)	2
Rheumatoid arthritis (714)	2
Relapsing polychondritis (710.8)	1
Raynaud's disease (443.0)	1
Niemann-Pick disease (272.7)	1
Immunodeficiency, congenital(279)	1
Sideroblastic anemia (285.0)	1
Myelofibrosis (789.8)	1
Metachromatic leukodystrophy (330.0)	1
Diabetic ketoacidosis (250.1)	1
Diabetes mellitus, not otherwise described (250)	1
Morbid obesity, death following revision of gastroplasty (278)	1
Aspiration of gastric contents, unexplained (E911)	1

\* Diabetes mellitus was listed a total of 52 times  
(6.3%) among 832 deaths (see text).

**Total 832 (99.8%)\***

+ rounding artifact



# QUANTITATIVE OBSERVATIONS OF HYMENS IN PREPUBESCENT FEMALES SELECTED FOR NON-ABUSE

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## ABSTRACT

The maximum hymenal opening was evaluated quantitatively in 111 prepubescent females during routine physical examinations in a pediatric subspecialty office. For comparison, an additional 53 females referred by child protective agencies were also examined. Analysis of data show "non-abused" groups may be separated statistically from "abused" groups on the basis of area of hymenal opening. The mean area for the "non-abused, non-masturbate" group was 6.4 mm<sup>2</sup>. The upper limit of area (mean + 3 S.D.) of hymenal opening in this group was 24.1 mm<sup>2</sup>. A child having a hymenal opening diameter of 6.94 mm or less has a 99% chance of being in the "non-abused" group. The area of hymenal opening for "non-abused" groups did not change with increasing age, height or weight. A skilled pediatrician knowledgeable in the area of sexual abuse may obtain clinically relevant information with ordinary office equipment and trained personnel. Regular and repeated observations of genitalia during routine health maintenance examinations are vital baseline measurements for the physical and mental health of young female patients.

## INTRODUCTION

Pediatricians are often called upon by courts and other authorities to render judgements regarding the possibility of sexual abuse in prepubertal patients based on physical examination. A total of 1587 cases of possible sexual abuse females age 13 & under were reported to the Alaska Division of Family and Youth Services (DFYS) statewide during 1987. Since Alaska's population of females of that age group was 68,993, the calculated incidence of possible sexual abuse would be 23/1000. Further inquiries of Alaska police departments revealed 346 cases had been investigated in 1989.

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When estranged parents accuse each other of sexually abusing their children, the physician is caught in the middle and needs objective criteria to determine whether the accusation has merit. Unfortunately, fourteen reports of significant physical findings in sexually abused females give conflicting criteria for excluding sexual abuse (Table 1).

Attorneys argue lack of physical evidence of sexual contact means that sexual contact did not occur. The landmark study by DeJong and Rose (1) demonstrated in cases of sexual abuse proven through the legal process of weighing all of the verbal and physical evidence by an impartial jury, physical evidence was present in only 23% of felony convictions. But what are the criteria for "physical evidence?" Measurements of "normal" openings in various studies (2-16) range from 4 to 10 mm. In none of these studies were children excluded who had risk factors for child abuse or sexual abuse. The present study attempts to refine these observations on a group of girls specifically selected for "non-abuse".

An elegant study by Emans et al. (2) demonstrated differences between girls with a history of sexual abuse, girls with other genital complaints, and girls whose genital examination was part of a routine health evaluation. Screening for factors associated with sexual abuse was not done on their control group. Considering the high prevalence of sexual abuse across all segments of the population (estimates are 15-25%) the control groups in Emans' study may contain girls who had been sexually abused.

McCann et al. (16) sampled children who were volunteers for "free research project examinations." Of the 114 subjects initially enrolled, seven were excluded because of onset of puberty and three were excluded because parents suspected abuse. The study was potentially flawed by the investigators' exclusion of 11 girls on the basis of the genital findings. The investigators believed the physical findings were indicative of child abuse. Thus, a circular argument was created - the criteria which McCann et al. wished to establish were used to define the study population. Eman's editorial

Table 1

## Published Values of Hymenal Opening

Author	Study Population	N	Diameters of Normal Hymen Opening in mm	
			Horizontal	Vertical
Emans et al	sexual abuse	119	4.4	4.2
	routine health exam	127	2.8	3.1
	genital complaints	59	5.1	5.0
Schubin	(not specified)	-	10.0	-
Muram	sexually abused	205	10.0	-
Herman-Giddons, et al	sexually abused	375	< 5.0	-
Cantwell	neglect only	157	< 4.0	-
	sexually abused	1000+	-	-
White	sexual contact	144	-	-
	at risk for sex	75	-	-
	friend's children	23	< 4.0	-
McCann	refer for sex abuse	172	-	-
	preschool	50	5.7	7.1
	early school age	52	5.9	6.7
	preadolescent	42	6.5	7.7

(15) on McCann's study note the need for a study in which independent criteria are used for defining the study population. The present study attempts to avoid this circular argument.

## RATIONALE

The purpose of the study is threefold:

- (1) To provide quantitative data on the area of the hymenal opening of females selected for non-abuse by independent criteria;
- (2) To determine whether area measurements have any predictive value in separating non-abused from sexually abused females;
- (3) To determine whether simple clinical techniques in the hands of knowledgeable pediatricians are sufficiently reliable for the evaluation of female genital structures. Note: the goal is not the establishment of diagnostic criteria of sexual abuse, which is a complex psychosocial disease.

## METHOD

This study consisted of 164 patients examined between June 1, 1988 and March, 1989. Entry of patients (N=164) into the study was via two routes: (A) "non-

abused" subjects, and (B) "referred" subjects. "Non-abused" subjects (n=111) were derived from a solo practice of general pediatrics and pediatric gastroenterology. Subjects were Caucasian, and from widely varying socioeconomic status. "Non-abused" subjects were prepubertal females who had come to the office for a routine health examination, and demonstrated no risk factors associated with abuse (16). Patients who had risk factors suggestive of sexual abuse were excluded from the study group. For example, subjects with parent(s) who gave unreliable or discrepant history in the past, dysfunctional families with frequent use of drop-off baby-sitters, so-

cial isolation, parent(s) with unrealistic expectations for the child, history of DFYS referral, addictive behavior, multiple intimate personal relationships, and/or live-in boyfriends who baby-sit were excluded. Children who also made statements regarding "bad touching" were also excluded from the study. Initial interviews were conducted by a nurse trained in the area of sexual abuse. Questions were also directed toward detecting the occurrence of nightmares, major changes in school behavior, etc., as well as any concerns the parent or guardian might have regarding the possibility of sexual abuse of the child. The "non-abused" subjects were further subdivided into two groups "non-abused non-masturbate" (n=100) and "non-abused masturbate" (n=11), on the basis of the parent(s)' observed knowledge of genital manipulation by the child. All data were obtained and recorded by a trained nurse to minimize bias by the clinician. The clinician was also blind to the historical information gathered by the nurse at the time the measurements of hymenal opening were made and recorded in the chart in the standard format (17).

Patients entering the study group via route B were referred by police or social workers from the Alaska Division of Family and Youth Services. They were also subdivided into two groups, pending outcome by the child abuse authorities: "abused" and "suspect". The "abused" group (n=37) consisted of children who had been judged by the court system to have been sexually



abused (sexual abuse does not necessarily mean penetration). The "suspect" group (n=16) consisted of the remaining children referred but for whom sufficient evidence was lacking for conviction, or the legal process was not complete at the time data collection was terminated. All children were examined by the pediatrician in the presence of their parent and/or a supportive adult, and the trained nurse. A complete physical exam was done, including all parts of the body, in a relaxed non-threatening manner. Less vulnerable parts of the body were examined first to gain the child's trust and confidence. Only the part of the body currently being examined was disrobed, and then redressed immediately after the examination of that part. When the child's vulvar structures were examined, she wore regular clothes from the waist up, was placed on the exam table in the dorsal lithotomy position, with an accompanying adult cuddling her upper part and maintaining eye contact. These techniques were used to achieve full relaxation and cooperation of the child, as relaxation can make the opening larger (18). The nurse's warm, reassuring personality and demeanor greatly facilitated the child's participation. The nurse instructed the child to draw her knees up with feet together near the buttocks, then letting the knees fall laterally "like butterfly wings". The examiner gently grasped the lower part of the labia majora and applied traction laterally and inferiorly. With this technique, air usually entered the vagina and full opening of the hymen was visible. Vertical and horizontal diameters were measured in reference to a calibrated scale: an ICM segment clipped from a plastic insertape was glued on the examiner's index fingernail and placed immediately adjacent to the hymenal opening. All measurements were repeated. Illumination was provided with a fiberoptic extension (Welch-Allyn part # 43300) on a 4.5 volt otoscope handle. To evaluate the reliability of the clinician's measurements, repeat hymenal measurements were made on 29 patients who had entered the study via route (A) "non-abused." The first 29 patients available for subsequent visit were used for a test of reliability of the techniques. To avoid bias, the clinician was unaware of the value of previous measurements until after he had examined the child, made the new measurements, and recorded them in the chart. At the time of the second visit, history from the parent indicated no new significant risk factors.

Because all the pediatrician actually sees is an orifice, the **area** of this opening (not the diameter) was chosen as the dependent variable. The space which is

unoccupied by tissue may have a variety of shapes (oval, round, crescentic, irregular, etc.). When the hymenal opening is inspected, the observer is first impressed with the magnitude of the **entire** area of the opening. Differences in area are obvious, whereas differences in diameter are more subtle (Figure 1).

The area of the hymenal opening was calculated using the formula for area of an ellipse since this formula is useful to approximate area for a variety of

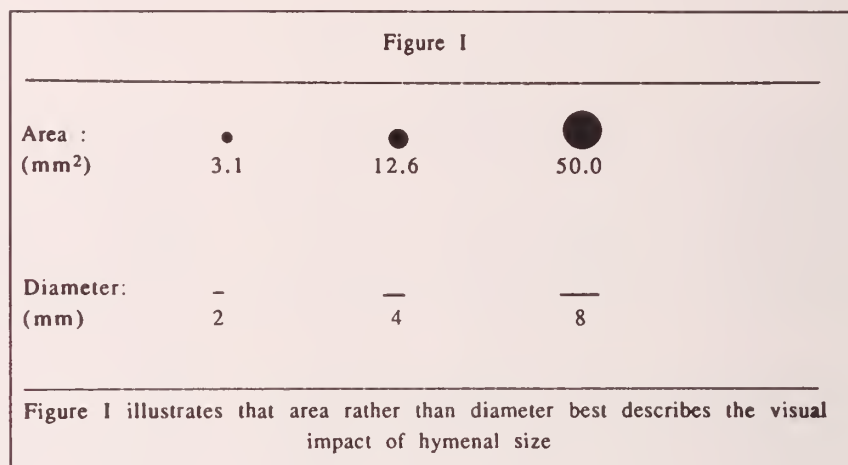


Table 2

**Comparison of Horizontal Diameter to Hymenal Opening**

Diameter	Area
2 mm	3.1 mm²
4 mm	12.6 mm²
6 mm	28.3 mm²
8 mm	50.2 mm²
10 mm	78.5 mm²

Table 3

**Age Classification and Frequency by month**

Class	Frequency	Age in months
1	15	1-24
3	42	25-48
5	46	49-72
7	36	73-96
9	14	97-120
13	11	121

shapes. See table 2 for quick comparison of areas to previously published data using horizontal diameter alone. The data were analyzed with the Statistical Package for Social Sciences (SPSSx) subroutines on the University of Alaska, Anchorage Vax computer system. Hymenal area was defined and equal to one half the horizontal measurement times one half the vertical diameter times pi ( $A = H/2 \times V/2 \times 3.14$ ). Measurement error was examined to establish reliability confidence intervals. Subjects' ages in months were assigned a two year range and grouped ordinarily by class and frequency (table 3).

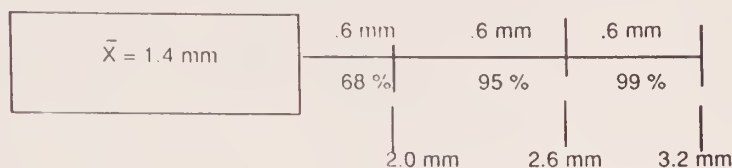
Several intercorrelation matrices for individual and group data were generated. Analysis of variance, Duncan post hoc analysis, crossbreak, and discriminant analysis were also conducted and specific relationships were examined for hymenal area, age, height, and weight by group. In addition, analysis of covariance with height and weight as covariates and discriminant analysis were used to determine statistical differences between groups and predict group membership class.

## RESULTS

The randomly selected sample of 29 subjects utilized for the repeat testing procedure helped establish the reliability of the measurement. The test-retest correlation coefficient was calculated to be  $r = 0.87$ . A further analysis using the pooled estimate of variance from each testing provided a Standard Error of Measure (S.E.M.) equal to 0.6 mm of diameter. Figure 2 graphically represents the measurement error, upper limit and degree of confidence. This means the "true" diameter of hymenal opening, when on a single measurement was recorded as 3 mm, could have been anywhere between 2.4 and 3.6 mm with a 68% confidence limit. To extend the confidence limit to 95%, the "true" diameter could fall between 1.8 mm and 4.2 mm. The data refer only to accuracy of measurement, and do not reflect variation within the study population.

The age classification ranges as shown in table 4 provided a revealing cross-tabulation of means and stan-

Fig. 2. Non-abused Sample Mean, Standard Error of Measurement, Upper limits, and Degree of Confidence



Conclusion: A child having a hymenal opening of 3.2 mm or less has a 99% chance of being a member of the non-abused group.

dard deviations of hymenal area by group. Means and standard deviations across "non-abused non-masturbate" group were found to be remarkably consistent, small, and restricted to a very narrow range. The mean area of hymenal opening was 6.4 mm<sup>2</sup>. (This is equivalent to a horizontal diameter of 2.9 mm). Because the

Table 4  
Means and Standard Deviations  
for Hymenal Area for Age by Group

	Age class						
	1	3	5	7	9	13	Total
<b>Group</b>							
Non-abused non-masturbate							
M	8.1	4.5	6.8	7.2	7.2	7.2	6.4
n	12	31	30	17	5	5	100
SD	11.8	3.4	5.8	3.9	5.4	3.6	5.9
Non-abused masturbate							
M	9.4	41.8	12.5	35.4	7.8		30.2
n	1	3	1	5	1		11
SD		20.1		22.8			21.6
Abused							
M	29.8	46.5	39.1	54.7	53.2	128.7	54.0
n	2	4	10	11	7	3	37
SD	28.8	16.3	21.6	21.0	16.8	48.7	32.1
Suspect							
M		46.3	41.0	34.8	50.2	72.3	51.2
n		3	4	3	1	5	16
SD		19.5	16.1	19.2		59.5	36.5



Table 5

**Hymenal Area Intercorrelations with Age,  
Height and Weight by Group**

Group	Age	Height	Weight
Non-abused (non-masturbate)	.08	.03	.08
Non-abused (masturbate)	-.03	-.21	-.21
Abused	.63*	.58*	.60*
Suspect	.67*	.60*	.74*
Total	.40*	.46*	.47*

\*  $p < .001$

observed areas are skewed toward the smaller end of the distribution curve, the standard deviation ( $5.9 \text{ mm}^2$ ) is nearly as large as the mean. The mean + 3 S.D. is  $24.1 \text{ mm}^2$ . Expressed in terms of diameter, the mean and standard deviation are  $2.86 \pm 1.36 \text{ mm}$ . The upper limit of hymenal diameter for (mean + 2 S.D.) is  $5.58 \text{ mm}$ ; and (mean + 3 S.D.) is  $6.94 \text{ mm}$ .

For comparison purposes, the mean hymenal area of the abused group is  $54.0 \text{ mm}^2$  with a S.D. of  $32.1 \text{ mm}^2$ . Again, to express these data in terms of horizontal diameter, the mean and S.D.'s are  $8.28 \pm 6.38 \text{ mm}$ . The variance in hymenal area comparing all four groups is considerable with remarkable overlap across groups as shown in figure 3.

However, regardless of the variance between groups, the area of hymenal opening did not increase significantly with age for both "non-abused" groups as depicted in table 5. Interestingly, early analyses for *all* groups combined indicated what one might expect, namely that hymenal area is likely to be influenced by the age, height and body weight of the child. These

findings were  $r=.40$ ,  $r=.46$  and  $r=.47$  respectively and all significant at  $p < .001$ .

Anatomically, hymenal area could vary across age groups due to natural maturational factors. Yet, further correlational analysis of the data by group indicated the hymenal opening was not significantly related to the growth of the rest of the body for "non-abused non-masturbates" or "non-abused masturbates". Thus, mean area of hymenal opening in non-abused groups were not statistically different from one age class to another. Significant individual group correlations for age by hymenal area was more frequent and consistently found for "abused" and "suspect" groups.

A Duncan post hoc analysis showed that "non-abused non-masturbate" was significantly different from the other three groups at the  $p < 0.05$  level (table 6). "Normal masturbate" was significantly different from "non-abused non-masturbate", "abused" and "suspect" groups, but the relatively small number of subjects ( $n=11$ ) does not allow for firm generalizations. The "abused" and "suspect" groups were not significantly different. Group separation showed, age, height and weight significantly related to hymenal area for abused and suspect subjects. These results may possibly suggest older females in the abused and suspect groups were more likely to have larger and more disproportional hymenal sizes, particularly concomitant with height and weight.

Alternatively, hymenal area in non-abused remained stable and was less influenced by age, height, or weight. Normal masturbate, abused, or suspect groups had substantial variations consistently across age groups. Analysis of variance revealed a significant difference between hymenal area  $F(1,160)=62.4, p < .001$  (table 7). Standard deviations suggest a common homogeneity of variance for normals regardless of age, while significant variations in hymenal areas were more common in the other groups. Larger fluctuations were particularly evident across all age groups in the "abused" and "suspect" groups.

Larger hymenal area values were common in comparison groups when matched with normals of the same age range. Correlation coefficients were obtained for actual age in months and not by class. These significant positive relationships for age and hymenal area suggest older females in this sample may have been more likely to have been abused and perhaps more than once.

A discriminate analysis was able to predict group membership with 84% confidence based on hymenal area alone. Further predicted group memberships are presented in table 8. An area of mean + 2 S.D. is strongly suggestive that the child has not been abused (confidence level = 94.7%). Future data may add to the consistency of these findings.

Table 6

**Duncans Post Hoc Analysis of Hymenal Area**

Group	1	2	3	4	Means
Non-abused (Non-masturbate)		*	*	*	12.8
Non-abused (masturbate)			*	*	60.5
Abused					102.4
Suspect					108.0

\* significant difference between groups  $p < .05$

Table 7  
Analysis of Variance

SOURCE	d.f.	sum of squares	mean square	F ratio
Between groups	3	305704	101901	62.42*
Within groups	160	261184	1632	
Total Variance	163	560889		

\*p.<.001

Preliminary indications suggest a rather reliable and stable hymenal area in "non-abused" prepubescent females across age with greater disparity for those who have been abused. This variance across groups, regardless of age, is most graphically demonstrated in the entire range for each group in figure 3.

## DISCUSSION

One may question the reliability of the measurement of hymenal opening. In the current study, the Standard Error of Measure was 0.6 mm. An individual measurement of diameter may be in error by  $\pm 1.2$  mm at the 95% confidence limit. Thus an experienced pediatrician does not need a colposcope to make reliable and clinically significant measurements of hymenal

opening. This study was specifically designed to evaluate hymenal openings quantitatively for females selected for non-abuse. Samples in other studies have primarily examined abused children. This study gives comparisons with females referred for abuse, but is not intended to provide definitive quantitative findings on abused females. The major importance of these data is the demonstration of the upper limit of normal in a clinically defined normal population. Further studies are needed with greater emphasis on independence of clinical ex-

aminations and group membership that also includes interjudge reliability of clinical observations.

The authors wish to emphasize the importance of physicians consistently evaluating the perineal area carefully and documenting observations, as components of the routine health assessment of females. The value of a single measurement is not as meaningful as a change in a value. Provided the examiner is skilled and exercises care to make reliable observations, baseline measurements of hymenal opening are vital medical data for every prepubescent female.

The remarkable similarity in size of the hymenal opening of the "non-masturbate" non-abused females across all age groups is similar to the observations of White et. al. (10). The hymenal opening of the older child was not larger than that of a preschool child. Our data show the vaginal orifice does increase considerably with age for only females in the abused and suspect groups. The opening in the non-injured hymen remains virtually stable and unchanged prior to the onset of puberty. Interpretation of the observation of females for possible sex abuse with these data in mind can enhance the accuracy of the diagnosis.

The selection process by the trained nurse for entry into normal groups was made by interview. Patients in the present study were excluded from non-abuse groups if risk factors for sexual exploitation were found. Despite these precautions, some children entering the study via route A may have been manipulated or sexually abused. The authors' attempt was to provide initial data on "non-abused" Caucasian females typical of the general pediatric practice. Errors induced by non-detection of abuse would tend to make observed openings larger.

To avoid circular logic, the group assignment was made independently by the courts and referral agencies rather than by the investigators. De Jong and Rose report physical evidence was present in only 23% of convictions for sexual abuse (1). The current report emphasizes the observation of non-abused females.

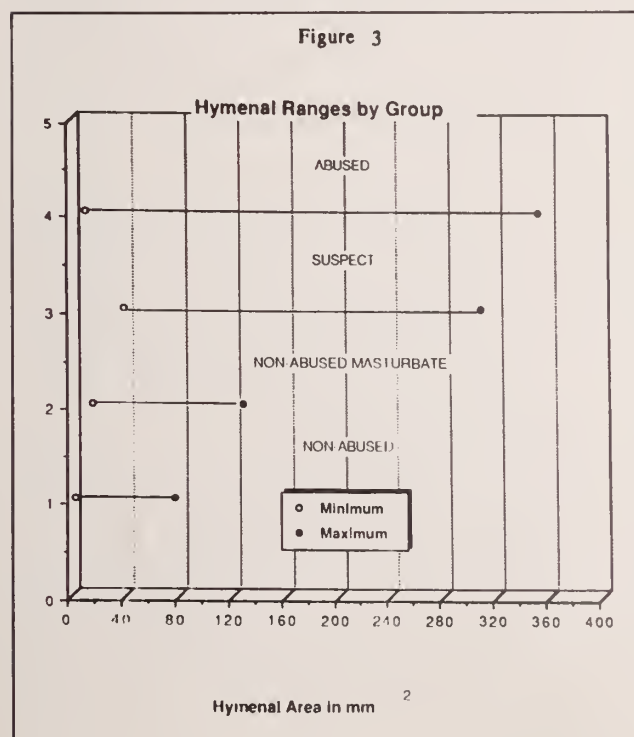




Table 8

**Discriminant Function Analysis of Predicted Group Membership**

<b>Actual Group</b>	Non-abused Non-masturbate	Non-abused Masturbate	Abused	Suspect
<b>Predicted Group</b>				
Non-abused non-masturbate	94.7%	4.3%	1.1%	0.0%
Non-abused masturbate	44.4%	33.3%	22.2%	11.1%
Abused	8.6%	22.9%	68.6%	17.1%
Suspect	6.3%	37.5%	56.3%	25.0%

Note: These data show the percent chance that a subject would be assigned to the correct clinical group by measurement alone. For example, 8.6% of subjects actually in the abused group will have measurements falling within the prediction for the non-abused non-masturbate group.

The data on the females with risk factors for sexual abuse are offered only as contrast, and are commensurate with the findings of Cantwell (7,8), Hermann-Giddeons et. al. (6), Pokorny, White et. al.(5), Emans et. al.(2), and McCann et. al. (16).

Some reports in the literature imply reliable measurements of size of hymenal opening are impossible. The present data demonstrates a trained and experienced observer can make reliable measurements, even without a culposcope. Clinicians may practice taking measurements repeatedly until sufficiently skilled in obtaining reproducible results.

Masturbation is another factor important for the examining physician to consider when evaluating females for possible sexual abuse. Other studies have not mentioned this factor. The present data indicate the openings in the hymen may be larger in these subjects, but more extensive research is needed to determine the range of hymenal opening in females who masturbate. Unusual genital care and hygiene practices may also induce changes in genital findings (13).

Patient relaxation is yet another important factor in observing the full opening of the hymen (18). In the present study, "non-abused" females appeared more relaxed than the "referred" females. Any bias in area of hymenal opening introduced by inadequate relaxation would tend to minimize differences between groups (relaxation leads to larger apparent opening). Despite potentially less relaxation in the females of the "abused" or "suspect" groups, their hymenal openings were larger.

The **DIAGNOSIS** of "vaginal penetration" must consider all sections outlined in the Position Statement of the Academy of Child and Adolescent Psychiatry (19), and must not be based entirely upon the area of hymenal opening. In the study by McCann et. al., the

vertical and horizontal trans-hymenal diameters were measured using a colposcope equipped for photography. Reliability estimates were made ( $r=.83$ ) which were comparable to our study. If one uses their data to calculate the area of hymenal area on girls examined in the same position as the current study (supine, traction), the average value would be approximately 24.6 mm<sup>2</sup>. Our average is 6.4 mm<sup>2</sup> for the non-abused non-masturbate group.

The commentary (15) on McCann's study (14) by recognized experts in the field also emphasized the need for measurements to be made on "non-abused", which were not done in the

McCann study. The present study provides these data.

Another study (12) comparing colposcopic and unaided observations reports no lesions seen through the colposcope that were not seen with the unaided eye. Even McCann (20) admits that colposcopes are expensive and difficult to use in the young child.

When are physicians' genital assessment techniques adequate to qualify them as "expert" observer? Clinicians need to make repeated observations until their test-retest measurements have a correlation coefficient greater than 0.8, whether they use the unaided eye or magnification instruments. It is not instrumentation that is at issue but rather the importance of reliable and valid observations during all routine examinations that is paramount.

## CONCLUSIONS

- (1) A lack of sexual penetration can be suggested but not proven by an area of hymenal opening of 24.1 mm<sup>2</sup> (mean + 3 S.D.). This corresponds to horizontal diameter of 6.9 mm. Such predictive value does not obviate or diminish the importance of other predictors of sexual penetration, such as psychological evidence, or other physical evidence such as tears, scars, etc.
- (2) Masturbation may influence the size of the area of hymenal opening. This needs to be evaluated further in studies of more subjects.
- (3) A skilled and knowledgeable pediatrician using ordinary office equipment and personnel can obtain extremely useful information that the hymenal structures are outside the limits of normal. (A colposcope is not necessary to make reliable quantitative observations).

- (4) The area of hymenal opening of "normal" females who do not masturbate is relatively stable with age prior to puberty.
- (5) Regular and repeated observation of the genitals during scheduled health maintenance examinations is an extremely important baseline to establish.

A study of 111 prepubertal female subjects provides useful information, but a larger sample is required to enhance the external validity for generalizations to the population. Pediatricians are encouraged to do similar studies on totally non-abused females of several races and ages. Careful documentation regarding the presence or absence of masturbation habits, genital hygiene methods, degree of relaxation, and other factors which can alter the appearance of the hymen should be recognized. We welcome other researchers who share similar interests to collaborate on future studies. Together these research efforts can facilitate the development of state and national databases.

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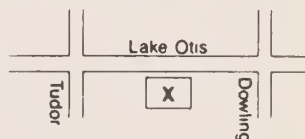
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# Homicide in Alaska Natives

by Paul A. Kettl, M.D.<sup>(1)</sup>

## SYNOPSIS

Homicide among America's minorities continues to be a pressing public health problem. In this study, we analyze the homicide rates for Alaska Natives (Eskimos, Aleuts, and Indians) for the three year period from 1982-1984 derived from a hand review of death certificates.

Homicide rates for Alaska Natives (24.9 per 100,000) are three times those for the United States for the same period (8.2 per 100,000). All age groups except the very young and the very old were more likely to be homicide victims, as homicide rates were remarkably consistent from age 15-74. Sixty-nine percent of homicide victims were male, and 58 percent were single. As with the rest of the U.S., death by gunshot was the most common method of homicide (in 46 percent of Alaska native deaths). More than half of the homicides occurred between 11 p.m. and 5 a.m. A slight excess of homicides occurred between June and August.

Homicide remains an important problem for Alaska Natives. Widespread availability of guns, and the continued modernization of Native cultures may be playing a role in fueling these high rates. Also, since such a large number of homicides occur in the early morning, alcohol abuse remains a suspect in contributing to many of the homicides. Much more work on minority homicide needs to be done to explore these and other aspects of homicide prevention.

Homicide is a neglected public health issue, especially for America's minority groups. In 1983, homicide was the 14th leading cause of death for whites, but was the 5th leading cause of death for African Americans and the 9th leading cause of death for people of other races (1). For African Americans, especially, homicide ends far too many young lives. One in 47 African Americans die by homicide compared to one in 240 whites (2). For males, the situation is even more bleak. One in 28 African American males will die of homicide. In fact, in New York City, homicide is the largest killer of men

between the ages of 16 and 45 (3). Between 1970 and 1983, homicide rates for African Americans were 6.7 times the rates for whites and 4.4 times the rates for other races (1).

Other minority groups also suffer from high homicide rates. While Hispanic homicide rates are lower than those for blacks, they are still disproportionately higher than rates for white Americans. In Los Angeles, the homicide rate for Hispanics was 2.3 times greater than for Anglos (4). Similarly, in an analysis of mortality data for five southwestern states from 1976-1980, the homicide rates for Hispanics was more than two and a half times the rates for whites (2).

Native Americans as a whole, also suffer from increased homicide rates which while higher than for whites, are lower than those for African Americans or Hispanics. In 1980, rates for Native Americans were 70 percent higher than for those of whites (1). Still, homicide for all Native Americans, and especially for Alaska Natives has been a vastly underresearched area.

The Alaska Native population consists of several ethnic groups. The term includes the Aleuts, who reside largely along the Aleutian chain; various Indian groups who inhabit the interior and southeast Alaska; the Inupiat Eskimos who inhabit the north and northwest of Alaska; and the Yupik Eskimos, who inhabit southwest Alaska. These peoples are culturally distinct with different histories and different languages. Although the ethnic groups are quite distinct, in this paper I will be forced to group them together because of the relatively small numbers of homicides involved. Insufficient data is available to distinguish between the various ethnic groups.

## METHOD

Death certificates for every Alaska Native who died from January 1982 through December 1984 were reviewed for cause of death. All death certificates that verified homicide as the manner of death were selected and their epidemiologic data reviewed. Specifically, sex, age, method of assault, marital status, hour of death and month of death of homicide victim were enumerated. Age and sex specific rates were then calculated.

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*This research was begun while Dr. Kettl was serving with the Indian Health Service in Alaska. He is now Assistant Professor of Psychiatry and Director of Psychiatry Residency Training at the Pennsylvania State University School of Medicine.*

While it is possible that death certificate data may underestimate the true incidence of homicide among Alaska natives, it remains the most reliable method available. During the study period, accidents were the foremost manner of death among Alaska Natives (5) and one must wonder if some of those deaths were, in reality, homicides.

Total Alaska Native homicide statistics from 1982-1984 were then compared to total U.S. homicide statistics for 1983 (5), and the differences between the two groups studied.

Because we are examining a relatively small number of deaths over a three year period, it is impossible to break down the data further into geographic or ethnic groups. I acknowledge that the high rate of homicide noted for the total Alaska Native population does not then imply that every Alaska Native ethnic group has a high homicide rate.

## RESULTS

Forty-eight Alaska Natives died by homicide in the three years studied as verified by death certificates. This produces a homicide rate overall for Alaska Natives of 24.9 per 100,000 population, three times greater than the homicide rate for the U.S. population, and substantially higher than the homicide rate for all Alaska residents (Table 1). Of course, the homicide deaths of Alaska Natives were included in the statewide homicide death rate. When homicide deaths are broken down by age, all groups between age 5 and 75 are substantially above both the U.S. total homicide rate and the rate for all Alaska citizens. Even more intriguing, however, is the distribution of homicide deaths among the various age groups. While in the U.S. population, homicide is

primarily a manner of death for adults aged 15-44, the homicide rate for Alaska Natives remains remarkably consistent from age 15 through 74. While homicide in Natives over age 75 did not occur during the study period, all other adult age groups continued to have homicide as a pressing public health problem (Table 1).

Alaska Native women were slightly more likely to be victims of homicide than women in the U.S. population. Thirty-one percent of Alaska Native homicide victims were women, compared to 23 percent of total U.S. homicide victims (Table 2). This difference, however, is small, and can easily be attributed to the small sample size in our study group.

Table 2.

### Proportion of Homicide Victims by Sex

	Alaska Native	U.S. Total <sup>14</sup>
Male	69%	77%
Female	31%	23%

When age differences in homicide rates are examined for both sexes, (Table 3) it is again remarkable how consistent homicide rates are virtually across all age groups for both sexes.

When the data is examined for marital status, it becomes clear that homicide among Alaska Natives occurs primarily among those who are single. Only 31 percent of homicide victims were married, while 58 percent of the victims were single. Only 4 percent of the homicide victims were divorced (Table 4).

The method of attack in Alaska Native homicide is virtually the same as that for the general U.S. population (Table 5). Death by gunshot accounts for almost half (46%) of homicide deaths while death from stab wounds is a distant second, representing 27 percent of Alaska Native homicides.

When we investigate the hour of death for Alaska Native homicide victims, it is immediately obvious that the hour of death is not random. The hour of death was only listed in death certificates for half of the homicide victims, but among those for whom the hour of death is known, slightly more than half of the victims died between midnight and 5 a.m. The remaining deaths are distributed in a largely random fashion throughout the rest of the day. This data must be interpreted carefully, however. First of all, the hour of death of fully half of the homicide victims is not known. Secondly, the hour of death is known, not the hour of attack. For example, one could have been attacked at one time of the day, and died

Table 1.

### Homicide Rates/100,000

Age	Alaska Native 1982-1984 3 yr. ave.	Alaska Total <sup>(5)</sup> Population (1983)	U.S. Total <sup>(5)</sup> Population (1983)
All	24.9	13.0	8.2
0-4	4.1	5.8	
5-14	4.9	1.2	
15-24	28.2	14.8	11.3
25-34	47.0	12.3	15.3
35-44	31.5	23.6	11.7
45-54	47.6	23.1	8.0
55-64	32.2	20.2	5.6
65-74	35.7	0	4.4
75-84	0	0	4.3
85+	0	0	5.6



Table 3.  
Alaska Native Homicide Rates/100,000 by Age  
and Sex for 1982-1984 (3 Year Average)

	Male	Female
0-4	0	8.5
5-14	4.1	4.3
15-24	35.0	22.6
25-34	73.5	20.1
35-44	52.9	9.6
45-54	69.2	29.5
55-64	19.1	45.6
65-74	70.9	0
75-84	0	0
85+	0	0

Table 4.  
Marital Status of Alaska Native Homicide Victims

Single	58%
Married	31%
Divorced	4%
Widowed	2%
Unknown	4%

Table 5.  
Method of Homicide Attack

	Alaska Native 1982-1984	U.S. Total <sup>14</sup> 1983
Gunshot Wound	46%	58%
Stab Wound	27%	22%
Impact Injury	23%	13%
Asphyxia	2%	3%
Hypothermia	2%	-

later. However, in spite of these cautions, the dramatic proportion of homicide deaths occurring in the early morning hours is indeed noteworthy (Table 6).

Finally, homicide deaths may show seasonal differences. Homicide occurs more often in summer than in winter. Twenty of the homicide deaths occurred during the four month period from May through August, while only ten occurred during the four month period of October through January. The highest suicide rate occurred during February, in which seven homicide deaths occurred. So while seasonal differences in homicide death were not dramatic homicide among Alaska Natives is slightly more likely to occur during the late spring and summer than the rest of the year. (Table 7)

Table 6.  
Hour of Homicide Death  
(known for 24 of 48 victims)

0:00 -	16.7%	12:00 -	0%
1:00 -	8.3%	13:00 -	0%
2:00 -	4.2%	14:00 -	0%
3:00 -	4.2%	15:00 -	4.2%
4:00 -	12.5%	16:00 -	4.2%
5:00 -	0%	17:00 -	4.2%
6:00 -	0%	18:00 -	4.2%
7:00 -	8.3%	19:00 -	8.3%
8:00 -	4.2%	20:00 -	0%
9:00 -	0%	21:00 -	4.2%
10:00 -	4.2%	22:00 -	0%
11:00 -	0%	23:00 -	8.3%

Table 7.  
Alaska Native Homicide  
Month of Homicide Attack

January	2.1%	July	10.4%
February	14.6%	August	12.5%
March	6.3%	September	8.3%
April	8.3%	October	4.2%
May	8.3%	November	6.3%
June	10.4%	December	8.3%

## DISCUSSION

Alaska Native homicide rates, hovering at three times the national average present a major public health concern. Although the rates presented in this paper are somewhat dated (1982-1984), they have stayed exactly the same since then. For Alaska Natives, homicide rates were 26.6/100,000 in 1985 (6), 20.8/100,000 in 1986 (7), and 22.9/100,000 in 1987 (7), giving a three year average of 23.4/100,000. These rates, remarkably similar to the three year rate presented here of 24.9/100,000 implies that the explosion of homicide continues in Alaska Native cultures. Equally troublesome is the fact that homicide rates are remarkably consistent across age groups. While the very young (those less than 14) and the very old (those older than 75) have low rates of victimization every other age group has at least triple the age matched homicide rates for the United States as a whole. In the rest of the country, homicide victims are primarily young, whereas, among Alaska Natives homicide is a problem for the entire age range. As in the rest of the United States, men are most commonly victimized by homicide, and single men seem to be especially at risk.

Almost half of all Alaska Native homicides result from gunshots. While the proportion of deaths from

gunshot is smaller for Alaska Natives than for the rest of the country, the sheer number of deaths remains a great concern. Because of the economic and cultural importance of subsistence hunting in the Bush, strict gun control would be politically and practically impossible in Alaska. Still, because almost half of the victims are killed with firearms, regulating handguns might help to reduce these rates. However, because the relative number of those who die by gunshot is lower among Alaska Natives than for the rest of the country, it is also clear that other factors must be taken into account in prevention efforts.

The timing of homicide deaths suggests another possible contributing cause. Among Alaska Natives, homicide deaths are most likely to occur in the early morning hours. Slightly more than half of the victims in our survey died between 11 p.m. and 5 a.m., the timing of these violent events in the early morning suggests that alcohol may play a role in many of the homicides. For example, in their survey of homicides for all races in New York City, Tardiff and Gross (8) found that the highest proportion of homicides occurred between 8 p.m. and 1 a.m. Reviewing the blood alcohol levels of these victims showed that 20.1 percent of male victims and 18.5 percent of the female victims had blood alcohol levels greater than 0.1 percent. A higher number, 38 percent of males and 36 percent of females had detectable blood alcohol levels not reaching the presumptive level for driving purposes. A similar study in Los Angeles produced similar results. 30.2 percent of homicide victims had blood alcohol levels higher than 0.1 percent and 46 percent had detectable levels of alcohol in their blood (4). While these studies are of homicide victims, not perpetrators, it remains clear that alcohol is an important contributing factor in many of the deaths.

With Alaska Native homicide occurring even later in the night, it remains likely that alcohol abuse would be a contributing factor. In the case of suicide among Alaska Natives it is clear that alcohol has a role to play in the genesis of those violent deaths. Fifty-four percent of Alaska Native suicide victims in the same period had alcohol levels greater than 0.1 percent (9), and Alaska Native suicide victims have a greater chance of having an alcohol abuse history than age and sex matched controls (10). This collection of evidence from homicides in other groups, and evidence of the involvement of alcohol abuse in suicide among Alaska Natives suggests that alcohol abuse and its social effects may play a role in the genesis of the high homicide rate evident in Alaska Natives.

While the highest frequency of homicides occurred in February, the three month period between June and August produced slightly more than expected homicides. This is compatible with Tardiff and Gross' (9) description of homicide death in New York, where

again June through August produced the highest rates. However, throughout the country as a whole, homicide rates are remarkably consistent from month to month (11).

Even with the data produced here, explaining the high homicide rate for Alaska Natives is not an easy task. Others have postulated reasons for high homicide rates for other culture groups, however, and their explanations may apply. Wolfgang (12) examined homicide rates in different developing countries and believed to the extent that countries with high homicide rates "westernize" without much turmoil, homicide rates can be expected to decline. However, if the acculturation of western attitudes is resisted, or leads to disintegration of or conflict within the norms of the existing culture, homicide rates, he believed, should increase. The Alaska Native cultures have been assailed by a barrage of factors over the last several decades. Along with economic growth may have come growth of homicide rates as an unfortunate side effect. Modification of traditional Alaska Native cultures by economic growth as well as the inundation of the culture by television and other media has been postulated to explain the increase in suicide rates (9,10,13). It is reasonable to assume that the same factors may be contributing to a homicide rate three times that for the rest of the country. Acculturation may well contribute to an increase in alcohol abuse, which may alter personal or cultural inhibitions against violence. Especially for the Eskimo peoples, who have always respected peace and non-violence, these high homicide rates require explanation. This acculturation perhaps fuels an increase in alcohol abuse which may be part of the answer.

Unfortunately, of course, the data in this paper raises more questions than it answers. Homicide was, and is, a large public health concern for Alaska Natives. It seems that gun control, alcohol education, and care for alcohol abuse might be appropriate public health issues to decrease these rates. However, more work on the subject must be done to prevent the loss of more Alaska Native lives through homicide.

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# Pediatric Lead Level Screening

by James W. Carpenter, M.D.<sup>(1)</sup>

## INTRODUCTION

Louis Sullivan (Department of Health and Human Services) stated in 1991 that "Lead poisoning . . . is the number one environmental threat to the health of children in the United States." Recently there is increased recognition of neurobehavioral effects and reduced IQ related to moderate lead exposures (Blood lead levels 10-25 micrograms per deciliter ( $\mu\text{g}/\text{dl}$ )).

## PURPOSE

In response to Centers for Disease Control (CDC) guidelines<sup>1</sup> the Department of Defense in late 1991 ordered that all military children be screened for lead exposure.

## METHODOLOGY

At Elmendorf Air Force Base Pediatric clinic, dependents under six years old are identified by questionnaire as low risk for lead exposure, or if their survey indicates possible risk factors for lead exposure a lead level is drawn.<sup>2</sup> At twelve-month well-child checks, a lead level is obtained on all infants. Specimens are sent to Smith Kline Beecham labs, and capillary specimens with elevated levels are repeated with confirmatory venous sampling.

## DATA

Between January 1992 and April 1993, 492 levels were drawn from children living in the Anchorage,

Elmendorf, Fort Richardson, Eagle River, Palmer and Wasilla area. The screening identified one child to have a lead level of 24  $\mu\text{g}/\text{dl}$ . Mother engaged in pottery as a hobby.<sup>3</sup> No home investigation was conducted and no other risk factors were identified. Subsequent testing showed levels of 18, then 2  $\mu\text{g}/\text{dl}$ . A second child had a level of 20  $\mu\text{g}/\text{dl}$  which was 15 upon venous confirmation. Another child had a level of 10  $\mu\text{g}/\text{dl}$ . All others were under 10  $\mu\text{g}/\text{dl}$ .

$2/492 > 10 \mu\text{g}/\text{dl} = 0.4\%$   
 $3/492 = 10 \mu\text{g}/\text{dl} = 0.6\%$   
 $487/492 < 10 \mu\text{g}/\text{dl} = 99\%$

## CONCLUSIONS:

This data does not represent a random sampling of South Central Alaska's children, but is a first step towards determining the risk of environmental lead in the Anchorage area. While only 0.2 percent of the area's children were screened, we did not find evidence of widespread lead exposure.

In addition to continued emphasis upon keeping lead out of the environment, we must identify risk factors for lead exposure. This demands epidemiologic investigation of all elevated lead levels so that we may keep safe our homes, workplace and entire community.


- 
1. CDC (Oct. 1991) Preventing of Lead Poisoning in Young Children
  2. Lead Exposure Risk Assessment Questionnaire
  3. Since 1990 all arts and crafts products must be labelled "caution" or "warning" if they have lead-based glaze, and they must include a list of ingredients and instructions for safe use by children. Unfired glaze may lead lead but is "food safe" if it has been correctly fired. MMWR 23 Oct. 1992, pg. 782. Vol. 41/No.42.

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(1) Headquarters 3rd Medical Center, 24800 Hospital Drive, Elmendorf Air Force Base, Alaska 99506-3700.

*The views expressed herein are the author's and do not reflect the official views of the Air Force or any government agency.*





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# ALASKA'S OWN FAMILY PRACTICE RESIDENCY PROGRAM

Barbara J. Doty, M.D.<sup>(1)</sup>

Health care in America is rapidly undergoing an evolution, and Alaska is not immune. Legislation to introduce comprehensive health care reform for Alaskans, including CHIPRA and others, is actively being pursued. Health care delivery in many Alaskan communities is undergoing transition. Boroughs such as Kenai are restructuring to develop area-wide comprehensive care packages. Hospitals and medical services in Bethel, Barrow, Kotzebue, Juneau and others are undergoing privatization through Public Law 638, and hospitals and physicians are forming partnerships throughout the state in response to increased demand for cost containment and comprehensive care.

A key component to health care reform, both nationally and locally, is an adequate supply of primary care providers, particularly family physicians, to serve as gatekeepers controlling the entrance point into the health care system and advocating for high quality, comprehensive care. Whether competitive managed care or universal care on a national basis, all proposals recognize one common denominator regarding health manpower - the critical role of primary care providers as the entry point to health services. Family physicians have proven themselves to be cost-effective in coordinating health care services, ordering appropriate tests, expediting specialist referrals, and efficiently utilizing hospital resources. As a result of their efficiency they are experiencing increasing demand by managed care recruiters as well as in traditional practice settings.

The supply of family physicians has not kept up with demand. There are over 400 family practice residency programs across the United States, producing approximately 2400 new family physicians yearly. These 2400 graduates must meet the attrition rate for the 68,000 family and general physicians currently in practice, as well as supply the increased demand for primary care doctors with the new health care delivery configuration. The existing supply of family physicians is aging, with 45 percent of family and general practitioners older than 55 years of age and 25 percent (17,000) expected to retire within the next five years. Alaska's figures are no better. The 180 private practice, 20 military, and 70 public health service family physicians serve a population of

550,000. Only 25 percent practice outside of Fairbanks and Anchorage, where 40 percent of the population resides. Those who do practice outside of the urban areas have greater demands on their services; they provide a higher proportion of total medical care as there are fewer specialists (Alaska has a total ratio of physician to population of 1 per 573 people as compared to the national average of 1 per 490). Over a third of Alaskan family physicians are commissioned in the Public Health Service or military and therefore only transiently based in Alaska. Their services are not accessible to the general public.

Both recruitment and retention of primary care physicians have been identified as problems for Alaska. Private practices report significant difficulties in recruiting physicians for their setting. According to a recent survey, the communities of Juneau, Palmer, Barrow, Nome, Valdez, Kodiak, Kenai, and Fairbanks report active efforts at recruiting over the past four years. The Public Health Service employs a full time recruiter to fill the expected 20 to 24 family practice positions vacated each year, a turnover of 30 percent. Statewide coordination of recruiting efforts and locum tenens opportunities was identified by the State Division of Public Health as a top priority in their recent report to the legislature. Relief physicians for solo or small group providers desiring a break from their hectic pace are extremely expensive and scarce. Obstacles to recruiting include isolation, high salary expectations, high malpractice costs, physician workload, and family lifestyle issues.

Alaska is one of only three states that does not offer a family practice training program. With respect to the other two states, Montana is actively pursuing a residency program and has offered third year rotations regularly on a semi-formal basis, New Hampshire works closely with the Dartmouth College-Affiliated Family Practice Program in Augusta, Maine. Other rural states such as Utah and Wyoming have two programs each, and Idaho, Hawaii and South Dakota have one each. New changes in health care policy have lead community hospitals and managed care groups to develop family practice residency programs of their own. The Accreditation Council of Graduate Medical Education (ACGME) reports at least 15 new programs currently under development. Studies have repeatedly shown that the location of residency training is a critical factor in influencing where a family physician graduate settles, with 70

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percent residing within a 100 mile radius of their training site. Community programs and those emphasizing rural training and experience have been more successful in getting their graduates to more remote settings than their university-centered counterparts. In other words, a program with a curriculum specifically focused on the needs and demands of rural practice is more likely to produce residents more comfortable in rural settings and who are more likely to choose rural practice sites.

Alaskan-based residency training offers a natural continuation in the training for Alaskan WAMI medical students, allowing them to transition back to their home state at the most crucial time in their decision-making on practice site selection. An absence of residency training opportunities is a frequent complaint of graduates of the WAMI program and of students who have had the opportunity to do clerkships in Alaska through the Indian Health Service or through private arrangements.

The time is ripe to develop a residency training program that considers Alaska's needs. Recognizing this, the UAA Biomedical Education Program (WAMI) in 1991 initiated a developmental project to explore the idea. Endorsements to the project have come readily from the Alaska Academy of Family Physicians, the State of Alaska House of Representatives, the Division of Public Health, and informally from the Alaska State Medical Association. A recent report to the Legislative Health Resources and Access Task Force from the State Division of Public Health recommended specifically that the State provide support for development of an Alaska-based family practice residency program. A steering committee of over 20 physicians representing the private, public, urban, and rural/remote sectors of Alaska convened in January 1992 to develop a consensus of what program format and curricula would best meet Alaska's needs. The following mission statement is the result of that meeting:

The mission of the Alaska Family Practice Residency Program is to supply Alaska with a work force of family physicians competent in the delivery of coordinated, top quality health care from the variety of Alaskan geographically remote and culturally unique settings. The program will serve as an educational center of excellence and clearinghouse for Alaskan physician recruitment, retention, re-education, and interaction.

A working group has been formed with leadership from representatives of WAMI, the Alaska Academy of Family Physicians, Providence Hospital, and the Anchorage Neighborhood Health Center, and consulting support from the Family Medicine Residency Network at the University of Washington. The group has been

meeting regularly and recently attended the annual Residency Assistance Program national meeting of the American Academy of Family Physicians headquarters and the annual spring meeting of the Society of Teachers of Family Medicine.

A working model for the residency project has emerged that consists of the following:

1. Family Practice Center (FPC)
  - located in Anchorage to serve as a hub for statewide educational interchange
  - functions as a self-standing clinic strategically located to service the urban medically underserved
  - staffed by the residency director, residents, and teaching faculty (required ratio of 1 FTE per 3 residents)
  - based on an ambulatory care clinic model
2. Hospital-based training
  - concentrated in first 15 months of curriculum
  - utilizes resources in Anchorage-based hospitals
  - meets inpatient training requirements in adult medicine, critical care, pediatric and newborn care, obstetrics, general surgery, emergency care
3. Didactic curriculum
  - lecture series held in conference format at FPC plus clinical conferences held regularly at Anchorage hospitals
  - networked teleconference via interactive video to access faculty and residents in regional sites
4. Rural practice training
  - on-site training at specific rural practice sites to be developed in Southeast, Interior, and Western Alaska where underserved populations exist
  - sites to offer inpatient care, obstetrics, emergency care
  - supervision to be supplied by an on-site coordinator who is part of a primary care group of at least 4 physicians who agree to serve as rural faculty
  - supplemental supervision to be supplied by on-site specialists, visiting consultants, mental health providers, and FPC faculty
  - curricula to focus on the special issues for successful rural practice, i.e. emergency stabilization and transport, cross-cultural medicine, behavioral medicine, substance abuse diagnosis and management, epidemiology and infection control, injury/accident prevention, occupational and

- industrial health, and health promotion/  
disease prevention
4. Subspecialty training
    - linear training and/or block selectives with specialists as required by the ACGME requirements. Specific number of contact hours for each subspecialty is designated. Specialties include Orthopaedics, ENT, Cardiology, Neurology, Ophthalmology, Dermatology, Urology, Neonatology
    - Elective options for additional training in specific areas

Evaluation of the quality of the training experience would be accomplished in a variety of ways, including in-house training exams, preceptor and faculty evaluations, self evaluations, and video critique. Graduates would be tracked through a standardized survey instrument such as the WAMI Network Resident Graduate Survey to assess outcomes in practice site selection and preparedness for rural family practice.

Funding sources identified for the residency program include Title 7 Graduate Medical Education Grants through the Bureau of Health Manpower, Community Health Center Linkage Grants, state legislative support, revenues from direct patient care, reimbursement through the Medicare Direct and Indirect Medical Education adjustments, and financial contributions from supporting hospitals and health corporations. Once programs are up and running, administrators with experience in residency program management report a decreasing dependence upon grant monies and a stabilization of revenues that typically results in the residency program becoming financially self-sufficient. The ACGME program certification specifically requires a balance in a program's emphasis between its educational mandate and its role in providing health care services; programs must reflect a solid educational commitment and cannot over utilize faculty and resident patient care revenues in their budget.

In summary, a family practice residency program in Alaska is not only feasible, it is a timely solution to an anticipated increasing shortage of primary care providers for rural Alaska. The process of residency program development is a long one, with a minimum 3 year process from conceptual development to full program accreditation and implementation. Commitment and active participation will be required of a host of health planners, hospital administrators, medical staff, legislators, and community leaders. The end result, however, will be a reliable, long term comprehensive solution to Alaska's physician manpower needs and an opportunity to establish a long-needed network between rural and urban providers for continuing education, recruiting, and information exchange.

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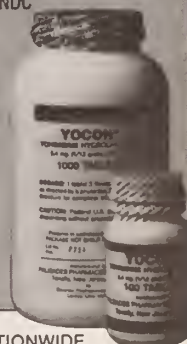
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# Sexually Speaking . . .

## The Darker Side of Summer

By Mary B. Cavalier, M.S.<sup>(1)</sup>

Each time this year I go into denial of the mind boggling number of tourists which flood our state. The traffic, the strange commercials, and the incredible questions like, "What time do the animals come out?"

Along with the increase of people, comes the increase of affairs of the heart, the "being swept off the feet by a stranger in the land of the midnight sun." These moments of passing passion also carry the increase risk of sexually transmitted diseases. Hence, the darker side of summer.

One of the major causes of misdiagnosis of sexually transmitted diseases are the paradigms of what type of people contract a STD. I know in my own practice that I don't hesitate to talk about safer sex and STDs with

someone in their twenties or thirties. But sit me in front of a 70-year-old woman who looks like my mother - well, that is another story.

We need to remember that STDs don't discriminate against sex, age, religion, race or sexual orientation. And that assumptions can carry with them serious medical complications. I would refer you to the June 1991, vol. 33, no. 2 issue of *Alaska Medicine* in which "Sexually Speaking" addresses an easy approach to taking a sexual history.

This article sounds more like a public health announcement than the usual tone of this feature. Well, without much more ado, watch the paradigms, keep the sunscreen and bug dope near by, and enjoy this magical land of the midnight sun!

<sup>(1)</sup> Robert Alberts, M.D. & Associates, 3340 Providence Drive, Anchorage, Alaska 99508.

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# Department of Health & Social Services

## Alaska Fetal Alcohol Syndrome Prevention Project



Dr. Peter Nakamura,  
Director Division of  
Public Health, Alaska  
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In 1991, the Alaska Department of Health and Social Services (DHSS) joined with the Alaska Area Native Health Service of the Indian Health Service (IHS) in signing Memoranda of Agreements (MOAs) with the National Centers for Disease Control and Prevention (CDC) to establish an Alaska Fetal Alcohol Syndrome Prevention Project. Through the Project the three signatories have agreed to collaborate in the development of model surveillance systems for fetal alcohol syndrome (FAS) and to generate epidemiologic data to support the development and evaluation of FAS prevention interventions.

Several DHSS divisions are participating in this endeavor: the Division of Alcoholism, the Division of Family Youth Services, the Division of Mental Health and Developmental Disabilities, and the Division of Public Health.

FAS is a leading preventable cause of birth defects and mental retardation in the United States. Features of FAS include prenatal or postnatal growth deficiency, abnormal facial features, and central nervous system (CNS) impairments. Surveillance of the condition, however, is complicated by five constraints: the difficulty of diagnosing the syndrome in the newborn; the subjective nature of the diagnosis; the variability in the severity of the conditions associated with the syndrome; age differences in the expression of the phenotype; and the lack of an ICD-9 code specific for FAS. Because of the constraints, there exists nationwide no reliable models for FAS surveillance.

The Project utilized several sources to compile a list of potential FAS cases, conducted a record review of those cases to generate a preliminary minimum FAS rate for Alaska, and then estimated the extent to which the preliminary data may have underascertained the true prevalence rate. The sources used included state data sources (birth and death certificates and medicaid claims), an IHS case file, and a pediatric practice case file.

In all, 348 potential cases were identified, and 323 records were reviewed using the following five criteria to verify case status: prenatal alcohol exposure or a maternal history of alcohol abuse, FAS noted by a physician, characteristic FAS facial features, CNS impairment, and growth deficits. Based on these preliminary data, the

minimum FAS prevalence for the State of Alaska was 0.5 cases per 1,000 live births during 1978-1991. For Alaska Natives, the minimum rate was 2.1 per 1,000 live births during the same period.

Eighty-three of the 323 met the five criteria and were considered to have FAS. Only 12 cases were identified by more than one data source. Seventy-five of the 83 cases were Alaska Native, a result which speaks to the extensive case finding activities of the Alaska Area Native Health Service. Since no previous work had been conducted to identify non-Native FAS cases in Alaska, Project staff confined its estimate of the underascertainment of the true prevalence rate to the Native cases.

Using a statistical technique called "capture-mark-capture" as it has recently been applied to epidemiologic surveillance data to estimate completeness of disease reporting, the Project reported an estimated maximum prevalence rate of 6.6 per 1,000 live births among Alaska Natives.

In addition to its surveillance activities, the Project is developing a profile of the mother who gives birth to an FAS child. Once we have a better understanding of the at-risk woman, we can then begin to track where she enters our health and social services system and target her for special interventions. In conjunction with this effort, the Project has conducted several surveys to assess the FAS-related knowledge, attitudes, beliefs, and behaviors (KABBs) of target audiences. To date statewide surveys have been conducted for women of child-bearing age, pediatricians, public health nurses, and the general public. The Project plans to survey ob-gyns during the summer. These surveys identify FAS-related strengths and weaknesses in the health and social services system and tell us where we need to allocate resources for education and services.

This Project is a major component of a CDC initiative to increase the nation's understanding of FAS. But it needs to be emphasized that FAS cases do not represent the entire spectrum of individuals affected adversely by alcohol. Fetal alcohol exposure may result in a wide range of adverse effects.

Our message is, there is no safe level of alcohol consumption for a pregnant woman.





## POSITION ANNOUNCEMENT

### Executive Director

#### POSITION

Executive Director of the International Union for Circumpolar Health (IUCH), a full-time position

The IUCH was established in 1981 with four adhering bodies:

The American Society for Circumpolar Health  
The Canadian Society for Circumpolar Health  
The Nordic Council for Arctic Medical Research  
The Russian Academy of Medical Sciences, Siberian Branch

The IUCH serves as an essential vehicle for international collaboration, cooperation, and communication between health scientists, practitioners, administrators, policy makers, and consumers in the circumpolar nations. According to its statutes, the objectives of the IUCH are:

- 1) To promote international cooperation in the study of circumpolar health;
- 2) To encourage and support research and exchange of scientific information in the circumpolar health sciences;
- 3) To promote public awareness of the current situation of circumpolar health;
- 4) To provide a means of communication with other organizations.

#### GOALS

The Executive Director of the IUCH will:

- 1) Ensure ongoing collaboration and communication of international scientists in circumpolar health;
- 2) Ensure the substantial involvement of aboriginal peoples from all circumpolar nations in circumpolar health issues;
- 3) Ensure the planning and implementation of successive circumpolar health congresses as the major vehicle of scientific communication. Such congresses will have an integrated indigenous peoples' issues component.

#### ACTIVITIES

The Executive Director of the IUCH will:

- 1) Maintain an office, membership list, filing system, and develop a newsletter;
- 2) Maintain financial records and prepare annual reports;
- 3) Develop and initiate a fund raising plan for the IUCH to allow for the sustained viability of the organization;
- 4) Liaise with national and regional governments in the circumpolar zone, as well as with international organizations such as the World Health Organization, the Inuit Circumpolar Conference, and the International Council for Scientific Unions;
- 5) Coordinate the annual meetings and other activities of the IUCH Council and its executive committee;
- 6) Develop and plan a series of international meetings, symposia, workshops, and seminars on specific circumpolar health issues;
- 7) Assist in the local organizing of the triennial International Congress for Circumpolar Health; including identifying funding sources, coordinating international delegates, involving indigenous organizations in the planning and delivery, coordinating international travel, and assuring the timely publication and distribution of the Congresses' proceedings. The tenth Congress will be held in Anchorage, Alaska, USA, in 1996.

The Executive Director of IUCH will report directly to the President of the Council of the IUCH. Day-to-day supervision will be in conjunction with the local representative and delegate of the IUCH. The position will be an annual appointment. The position and location of the office will be reviewed after three years. The position will be established as a contract for a self-employed individual.

**LOCATION**

Anchorage, Alaska, USA

**SALARY**

Salary is competitive and commensurate with experience and related to the job requirements of an administrative position.

**SEARCH**

Review of applications will begin on June 30, 1993 and continue until the appointment is made.

**QUALIFICATIONS**

- \* A college or university degree
- \* Demonstrated success in writing and obtaining scientific grants and general fund raising ability and experience
- \* Management experience in planning conferences and other administrative duties
- \* Knowledge of circumpolar health issues is essential
- \* Knowledge of computers is desirable
- \* Ability to travel on an international basis regularly
- \* Knowledge of international relations in Arctic and Antarctic regions
- \* Refined interpersonal skills will be required
- \* English is the official language of the Union and therefore all candidates must demonstrate a high level of both written and oral communication skills. Bilingual abilities for circumpolar peoples will be advantageous
- \* Cross-cultural experience with circumpolar indigenous peoples is essential

Please send a cover letter and resume to:

International Union for Circumpolar Health Secretariat  
P.O. Box 141594  
Anchorage, Alaska 99514





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The Committee is **NONDISCIPLINARY** and information is kept **CONFIDENTIAL**. If you, a colleague, or a family member is in need of our services call (907) 562-262. Ask for Pam Ventgen, or write: Impaired Physicians Committee, 4107 Laurel Street, Anchorage, AK 99508.

**HELP IS OUR BUSINESS!**



# Press "1" If You Hate Our Phone System

by David Karp<sup>(1)</sup>

*"Welcome to the Heartfelt Medical Group. You can dial the extension of any of the humans who work here or, if you do not know the extension, you can press 1 on your touch tone phone to reach the billing office; press 2 to make an appointment; press 3 to cancel an appointment; press 4 to leave a message for one of our doctors; press 5 for prescription refills; press 6 to speak to the insurance clerk; press 7 for a listing of all 250 of our telephone extensions. If the extension you reach is busy, please stay on the line and we'll let you listen to some very annoying elevator music. This message will be repeated every 20 seconds to remind you that we really care about your call."*

The use of computers in medicine continues to grow at an astonishing pace. Hospitals, physicians' offices, outpatient centers and research facilities use computers to assist in diagnosis and surgery, for patient scheduling, call back notification, medical record documentation and medication monitoring. Computers used in research are credited with expediting numerous medical breakthroughs, including cures and treatment of disease.

## **AUTOMATED PHONE SYSTEMS: PROGRESS OR AN ELECTRONIC POKE IN THE EYE?**

One use of computers that is beginning to have an impact on doctor-patient relations is the "automated routing unit" (ARU) telephone system that some hospitals, medical offices, and commercial businesses have installed to manage heavy telephone traffic. Typically, the telephone is answered by a recorded voice which identifies departments the caller can access by pressing a number. Some systems have "electronic mail boxes" into which one can leave a message. In some ARU systems, selecting any of the numbered choices gives the caller a second menu of choices, and sometimes a third or fourth menu. For example, the electronic voice at one large medical center directs callers to press #3 to reach the business office. After pressing #3, the caller is offered another set of numbers to choose "billing problems," "account inquiries," "errors," "insurance information," and so on.

A local bank's ARU plays a long self-congratulatory introductory message, followed by a commercial about the bank's current interest rates, loan opportunities, and branch offices. Finally, the caller hears a menu

selection. To find out if a check has cleared, for example, a caller works through three menus and then is told to enter his checking account number, social security number, and the number of the last check written. Make a mistake in entering any of the nearly 20 digits requested and the ARU reports, "you have entered an invalid account number," and instructs the caller to press yet another number to re-start the sequence. For some, opening an account at a different bank may be preferable.

Vendors of automated telephone routing units extol the ease with which calls can be directed to specific extensions or departments, thus minimizing use of staff and reducing personnel costs. One northern California hospital that plans to install the computer operated system expects to reduce overhead by phasing out its entire staff of live operators. ARU vendors insist that proper planning and design can reduce or eliminate delays and caller dissatisfaction. "If there aren't enough incoming lines and people to answer them," one representative said, "no automated system is going to work efficiently or be accepted by the public."

## **BUSY SIGNALS AND UNANSWERED CALLS COST THE CALLER**

Many callers connected to electronic routing phone systems complain that these systems are impersonal and unforgiving. Selecting the wrong extension or reaching one that is in use may terminate the call or play a busy signal until the caller hangs up and re-dials. The ARU in a San Francisco physician's office gives the following instructions: "To make an appointment press 1 now; for lab results press 2 now, for a mammogram appointment press 3 now [one wonders about the reaction of patients who pressed #1 "now" for an appointment as instructed, only to find that the person or machine at that extension doesn't schedule mammograms]; for the billing office press 4 now; and [last on the menu] if this is an emergency press 5 now." When we called this office, our selected extension was busy and unreachable during the next three hours. When we did not get a busy signal, we were placed on hold. After a 13 minute and a 28 minute hold, we gave up. The "emergency" line, which we tried as a last resort, remained busy.

Callers from other area codes who reach a busy ARU extension may not be aware that toll charges begin when the ARU first answers their call. One irate senior citizen wrote to her physician to complain that she not only had to listen to loud radio rap music which was playing while

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(1) Professional liability claims consultant, San Rafael, CA. Reprinted with permission.

she was on hold for nearly ten minutes, but she had to pay toll charges for the call.

## REACH OUT AND MAKE SOMEONE ANGRY

The most frequent complaints about computerized telephone answering systems, according to telephone company representatives, center on the systems' impersonal nature, the difficulty many have in remembering or following instructions, and frustration in not being able to speak to a person to ask for assistance before being placed into the electronic maze. "In hospitals, large medical offices and other businesses with many departments," one representative said, "the caller may be confronted with too many choices. He may be told by a recorded message to press up to ten numbers. By the time the third or fourth choice is mentioned, many callers get confused and lose track. Unless the system permits a return to the main directory list, the discouraged caller may just hand up." But even a return to the main menu can be frustrating, as one must listen to the entire introductory message again. Busy signals are another common irritant, as are unanswered calls from which there is no return. The caller's ire is heightened when, on repeatedly calling back to a previously busy or unanswered extension, he is told that the party he is trying to reach is "not in today." In a non-automated system, the caller would have learned this from the first live person who answered the phone.

Personnel at some facilities that use ARUs do not like the phone systems any more than their patients do. Several told us they receive many negative comments about their phone system. "We have to spend half our day apologizing to people who hate our phone system. Some patients are angry at us, and we have to spend even more time calming them down to find out what their medical problem is. Write a letter and complain," they advise callers. And apparently many people do. A cardiologist admitted that several colleagues have sent letters to complain they were unable to reach the office to arrange referrals. One referring physician wrote that he cannot afford to have an aide wait on hold while her call works its way up a queue of unknown length. The cardiologist is considering a return to "an old-fashioned multi-line phone system" staffed by an efficient receptionist.

Medical offices and hospitals that are considering computer-aided telephone routing systems might take note of studies that indicate many intelligent, educated, and clear-headed people already are intimidated by relatively simple electronic devices, such as videocassette recorders, automated bank tellers, and the benign and now old-fashioned telephone answering machine. Confrontation with one more impersonal electronic device—particularly when one is not feeling well—does little to

promote good will. Medical management and malpractice liability experts insist that doctor-patient relationships need **more** personal attention than they are currently receiving, not less. It is counterproductive, they say, to depersonalize a medical practice or hospital by using an automated phone system in which it takes too long before callers are greeted by a friendly person who can answer questions and offer assistance.

Medical facilities contemplating a change to an ARU might consider the medical-legal consequences of inordinate delays or confusion callers experience when they need urgent assistance—or think they do. Consider the anxious parent who calls the pediatricians's office for advice about her child's rising temperature, but cannot get through because the single advice line is busy; consider the patient in labor who gets trapped in her obstetricians's complex phone system; or the elderly patient who is disoriented and needs advice because he took too much medication. How long should these callers persevere before going to an emergency department or calling "9-1-1"? In an ordinary medical office phone system, an aide trained to assess the urgency of these calls can take a message or get prompt advice from a physician. Competent aides who answer telephones in a medical facility can reduce the callers' anxiety by assuring them their questions or message will receive someone's prompt attention. By contrast, few ARU system recordings tell callers how long they will be stranded on hold, how available people are at the other end, or how soon someone is likely to retrieve his or her electronic mailbox messages.

Facilities that have an automated routing phone system are encouraged to poll patients for their reaction and constructive suggestions. After such a survey, one clinic re-designed its main ARU menu and added additional phone lines for those departments which patients said were the most difficult to reach. But personnel in several other facilities we contacted praised their ARU and were certain callers will eventually get used to them.

No one should stand in the way of progress. However, if ARUs are to be a permanent part of our culture, but do not become more "user friendly," the public may clamor for an additional menu selection: "**Press 1 if you hate this phone system,**" followed by a recorded list of competitors who still value the personal touch.

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THE COMMITMENT CONTINUES

# History of Medicine in Alaska

## R. Holmes Johnson

"Dr. Bob", he is called. Robert Holmes Johnson, the son of the late Dr. A. Holmes Johnson and Fostina Bishop Johnson, was born in St. Helens, Oregon. At the age of four, the family moved to Portland where Bob attended elementary school. In 1938 they moved to Kodiak, Alaska where his father became the sole physician on the island. In order to squeak into high school, Bob crammed one year of algebra into two months.

There were twelve Caucasian families in the town of Kodiak and there was only one other Caucasian in his class. He was only twelve when he entered high school, but he was valedictorian when the class of eleven, the largest ever, graduated in 1942.

Since Bob was too young at sixteen to enter the military he went to Willamette University. Because of his interest in music, he ascertained who among his classmates could play instruments. This led to the formation of a fifteen piece swing band, the first of many he was to organize. But he neglected his studies and was placed on probation.

In April 1944 he was appointed to the United States Merchant Marine Cadet Corps, and at the San Mateo Basic Training School in California became a company commander. Considering the number of men lost during World War II in the Merchant Marines, this was a courageous thing to do. After six months sea duty in the South Pacific, where on the light side he found chasing sea turtles fun, he entered Kings Point Academy in New York where he was appointed to the regimental staff. He became a member of the Academy 'pit' orchestra as well as playing piano with a small dance band. He earned a third mate's ticket and a B.S. Before discharge in 1946, he made one trip to England. He liked the Merchant Marine, finding it a phenomenal admixture of people.

He returned to Willamette where he took eighteen hours of courses with five labs. He continued to play in bands, forming his own jazz combo. The combo did one nine week stint at a local night club, playing for four hours, six nights a week. He was elected to Alpha Phi Omega in his junior year and was president of his senior class.

After receiving a B.A. in biology in 1950, he was not accepted at medical school. He became a graduate student in human genetics at the University of Michigan, earning an M.A. in zoology and was finally accepted at the University of Washington Medical School. He became involved in a small dance band and was placed on probation at the end of the first year. He reestablished

himself by passing an examination in laboratory medicine.

In 1952 he married Marian James of Seattle. Their first child, Craig, was born in 1954 six weeks before Bob received his M.D. They drove to New York where Dr. R. Holmes Johnson interned at Brooklyn Methodist Hospital where his parents had met.

The year in Brooklyn was an experience since Bob made only \$50 a month. He was named Intern of the Year. On the return to Kodiak, the family drove across the United States and up the Alaska Highway.

Dr. Bob began practice with his father, Dr. A. Holmes Johnson. While Dr. A. Holmes had seen to the building of a twelve bed hospital, there was neither lab nor x-ray, therefore making diagnoses based on the history, physical examination and good judgement a challenge. Dr. Bob gravitated to his favorite discipline, obstetrics. He is most proud of the patient who had had a Cesarean section but whom he skillfully guided through vaginal delivery.

When the only doctor in Kodiak who performed abortions left, Bob negotiated a malpractice insurance rate that would permit him to do abortions. His record is good, because he carefully questions the patient's decision, provides a complete explanation of the procedure and risks, and follows up two weeks later. His experience belies most of the claims of the "right-to-lifers." Sometime he intends to write on this subject.

He is a doctor who makes house calls on alcoholics. He helped to establish the Alcohol/Drug Treatment program and the Mental Health Center in Kodiak.

He joined the American and Alaska Academies of General Practice and served as president of the Alaska Academy. He has been active in the Alaska State Medical Association, serving on the council for ten years.

Because he is talented in so many ways, one is moved to inquire as to why he did not make a career in music. He replies that his parents imbued him with the concept that one should make a contribution to the fulfillment of needs of other human beings. This made him choose medicine as his vocation leaving the pleasure of music and acting to a fulfilling avocation.

Like his parents, he has been involved in most of the civic and artistic happenings in Kodiak. Specifically he founded the Toastmasters Club and Kodiak-Baranov Productions, which has produced the "Cry of the Wild Ram." Frank Brink's epic, for 26 years. He has twice been named the best supporting actor in this production.

(continued on page 188)



# From Out of the Past . . .

## Medical Practice in Western Alaska Around 1900

by J. H. Romig, M.D.

(Reprinted from Providence Hospital Edition, Anchorage Daily Times, Thursday June 29, 1939)

### INTRODUCTION

Charles E. Chenoweth, M.D.



*Joseph H. Romig, M.D.*

When one reviews the history of medicine in Alaska, especially in the Western and Central areas, one name stands out more prominently, perhaps, than any other and that name is Joseph H. Romig.

Dr. Romig came to Alaska in 1896 as a medical missionary for the Moravian Church and began his work in their mission at Bethel. For seven years he labored at this mission.

He then decided to return to the states and established a practice in San Francisco. He had not been there long when the disastrous earthquake occurred in 1906 and he lost all of his equipment.

He was then offered a position as medical supervisor for the canneries at Nushagak. This he accepted and returned to Alaska. While at Nushagak, he was appointed Commissioner by Judge Wickersham and remained there for two years. In 1909 the Bureau of Education at Washington appointed him superintendent of schools and physician to the southwestern Alaska natives. While in the Indian Service he was headquartered at Seward from 1918 to 1922. Later he joined the medical staff of the Alaska Northern Railway and was stationed at Nenana in 1922-1923 and at Fairbanks from 1923 to 1930. From 1930 to 1937, when he retired, he was Chief Surgeon for the railroad residing in Anchorage. The following is his own story of his experiences before hospitals were established in Alaska.

Before 1896, there were but few white residents in western Alaska and they were composed of traders, a few missionaries and also a few teachers. The total white population was about 300, and the native people were living in their original primitive ways.

Among the Eskimo population of the Bering Sea region, the belief was that sickness was caused by the anger of some spirit, and disease was treated by Shamans, or medicine men, who attempted to appease the wrath of the offended spirit, and thus allow the afflicted to get well.

Early in the history of the Bethel Mission, a chieftain of the Mountain village became a convert. Later he became insane. The ravings of this chief were mostly about religion. They sent for the missionary to take the spell off of him and to entreat his God to restore the chief, something like they would do to have the Spirit appeased by their medicine men. This failed and on the following day the villagers tried to kill the missionary, but he escaped with a friendly stranger.

Two brothers of the afflicted chief took him out in front of all the people, stabbed him, threw him down and

set the dogs on him. He was killed in front of all the villagers, to warn them not to get religion.

### BETHEL MISSION FIRST POST

Six years after a full college course in medicine, and especially surgery, I arrived in Alaska. My duties were to change the belief of the people in the cause and cure of sickness. I was the first resident physician and surgeon to live in southwestern Alaska.

About the end of July 1896, I was standing on the bank of the river at Bethel. Behind me was the first office and hospital in Alaska west of Sitka. It was a small log building divided into two rooms. In the one were two wooden beds, and homemade furniture. In the other was a well supplied office with a homemade examination and operating table and basic stands.

### MAKES FRIEND OF CHIEF

Out in the river were many canoes and skin boats. The Eskimos were trying to harpoon a white whale.

Finally three canoes were seen to be fastened together and the three made for shore. In the middle boat sat a man who was not paddling. When the party reached shore, a well-built man of about 35 years came up on the bank holding his hand, which was bleeding freely. He had been hit by a harpoon and was seriously injured.

Cleansing, ligatures and sutures soon made a good looking hand out of a bad one. The man said little, but was very grateful. It was the first visit to the mission since he had tried to kill the missionary and had killed his brother for getting insane over religion. I do not know if he ever got religion, but he was a staunch friend thereafter. As he was then chief of his village, he had me adopted into his family of the big stomachs, and made me a coat with the family trimmings on it.

## OPERATES IN ODD PLACES

After this the sick and injured came from afar. I traveled hundreds of miles by dog sled each winter to give relief to those who could not come to the mission. Operations were performed in igloos, kachimas, bath-houses, at trading stations and missions. Sometimes messengers would travel two or three hundred miles or more to call for help, requiring a week of hard driving to answer the call and another week for the doctor to get to the patient, if he happened to be at home, and longer if he was away at the time of the call.

One year after a severe spell at Christmas time, a call came for aid for a man with both hands and feet frozen at Goodnews Bay. When nearly there, after two days of hard driving, with a dog team, a second messenger was met, who said the man had died. Returning home a message was awaiting from the Holy Cross mission, saying that one of the Fathers had suffered from the cold, and had a frozen foot, to come at once.

When 30 miles upstream, another messenger traveling by dog team was met, saying a trader from the Yukon was coming with a frozen foot, to hurry towards him as he was afraid of blood poisoning. It was necessary to return that night for more ether and supplies. On the second day, at noon, some 80 miles up the river in an Indian bathhouse, the trader was operated on. On a slab on the floor I gave him the ether, and the guide kept it up. Beside him were two native wooden bowls with boiled sterile solution and two candles. The man with the ether was scared, eased up the ether and the patient was sailing around Cape Horn in rough weather, using rough language. He said, just as the operation was finished: "Oh, etherize me with a brick."

He was afraid to be left behind, so as soon as he was out sufficiently to be put in a sleigh, the trip proceeded up river.

## MEETS BISHOP CRIMONT

The next day on the tundra land we sighted another dog team, whose message was to hurry to the mission as the Father was worse. We left the first case at Pinmuit on the Yukon with Mr. Tucker and went to the mission. The Father recovered and it was at this time that I met Bishop Crimont, and formed a friendship that has lasted 40 years, growing deeper as from time to time we met and had much in common for those in need and sickness.

On the homeward trip the first man was dressed at Mr. Tucker's place, and just as we were eating supper, a long team came across the Yukon heading for the cabin. A messenger had been sent to Bethel for the doctor to go to the Russian mission to see the trader's son who was seriously ill. Another team was sent out when they learned where the doctor was.

The next day, 50 miles down the Yukon, the trader's son was treated and then, over the low wasteland in a severe, drifting storm, the homeward journey was made. There were 700 miles of forced driving on this trip of 17 days, and not all good weather. There were many more trips and many heroic efforts to get the sick and injured to the doctor.

## ENTERS GOVERNMENT SERVICE

In 1909 I went into the Indian Bureau service and traveled from Yakutat to the mouth of the Kuskokwim, thousands of miles of winter and summer boating. I operated in the small hospitals then established at Cordova, Valdez, Seward and Nushagak and in all the schools and missions along the coast. The need of better hospital facilities was everywhere apparent.

When the U.S. Railroad was started, I entered its service and after 20 years retired as chief surgeon.

Colonel Ohlson was very ready and willing to improve the medical service. Much credit should be given him for it was under his advice and consent that I made two trips to Fairbanks to urge the Sisters of Charity to take on hospital work at Anchorage.

The dog team days were over. Messengers driving by day and by night no longer rush for the doctor. The wireless does that.

There are no more early and late trips in the cold and storm for the doctor to go hundreds of miles to relieve suffering, for now ambulances of the air --the airplane-- easily and quickly and safely bring the sick to the hospital.

The miner, prospector, fisherman and trapper, as well as the settler, has an assurance of care, should sickness or accident befall him. The settler can now safely bring his family to this land, knowing that there is a hospital of the best, modern style, ready and willing to care for him or his.

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(continued on page 188)



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## Dr. J. H. Romig's Successors on the Yukon

After Dr. Romig's time in Bethel, there were no resident physicians in the Kuskokwim-Yukon Delta area for some years. Eventually the government sent Mrs. Herron, a public health nurse into the area based at Bethel and she served in this capacity for many years. When the airplane became available, patients would fairly often go to Anchorage or Fairbanks for care, paying a one-way fare of \$500 per person.

About 1930, the Alaska Native Service opened a hospital at Akiak, then the center of the reindeer industry. Dr. George and a small staff worked there until it was closed in 1934. The next hospital was placed at Mountain Village on the lower Yukon and was open for only a very brief period, apparently because the land it was built on was being eroded by the river so badly that it was in danger of falling in. At least this is the story told by those who live along the rivers. The doctor there apparently did a good deal of surgery.

The first hospital built in Bethel was opened in 1940 by the Alaska Native Service. It had about 35 beds and was served during the next ten years by Drs. White, Langsam (who later went to Nome), Albitre and Chalmers, and perhaps another one or two. Dr. Langsam was, and still is, referred to as the talking doctor by the Kuskokwim residents because he was the only doctor who has really learned to speak the language of the area.

When the hospital burned in 1950, in addition to the usual run of patients there were nearly 20 who had fairly recently been operated on for orthopedic problems by Dr. Philip Moore in one of his visits to the area. Their evacuation was completed without any loss or injury.

A two quonset emergency unit bulged at the seams until the present hospital was finished in 1954 and it has usually bulged at the seams ever since. The Alaska Native Service and later the United States Public Health Service has gradually increased the medical staff there from one in 1954 to four currently. In 1959 a physician started private practice in Bethel. It might be added that there are now about five nursing districts in Mrs. Heron's old area.

Harriet Jackson Schirmer, M.D.  
Bethel, Alaska

(continued from page 181)

The Johnsons live in a custom-built house which stands on a bluff above the sea. Marian Johnson was there alone with three children, one six weeks old, when the tsunami created by the Good Friday Earthquake of 1964 occurred. After this terrifying experience, they were without running water for a year. In 1965 Dr. Bob practiced alone for eleven months in a community of 4000.

Now, with the four Johnson sons grown, Marian is Director of the Baranov Museum. Kodiak is proud of its heritage and the museum is a busy place.

It is not surprising that the Alaska State Medical Association added to the many awards to Dr. R. Holmes Johnson, the A.H. Robins Community Service Award for 1992.

Gwynneth Gminder Wilson  
Auxiliary

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## A look at hospital care in New Archangel (1835-1840)

A previous article in this series briefly described the medical care available to the workers of the Russian-American Company beginning in 1820. The initial eight hospital beds had to be increased during the first decade to more than twenty, to accommodate the expansion of the commercial operations at the capital. Baron von Wrangell, one of the Russian governors, noted in the early 1830s that the hospital was a "large, well-maintained building that was generously supplied with all the necessities."<sup>1</sup> Whenever possible the company employed two physicians at the hospital, but recruitment difficulties precluded such a luxury most of the time. Much of the routine medical work instead was carried out by *feldshers*, almost a type of physician's assistant, usually mixed Russian-Aleuts who had been trained at the hospital.

Many of the doctors and *feldshers* who served in the early years have left little trace, a notable exception being Dr. Blaschke, introduced in the previous article. Within a month of his arrival, as mentioned, the devastating smallpox epidemic had absorbed all his energies. The widespread sickness in the capital made it necessary for him not only to make use of every available room at the hospital, but also to establish an additional small facility for women a short distance from the main building. Yet, a visitor--Lieutenant Beleher of H.M.S. *Sulphur*--remarked in 1837, perhaps near the peak of the epidemic, that the hospital was "comparatively clean" and comfortable. He was especially impressed by the practice of placing a sign over each patient's bed giving his name, date of admission, and diagnosis, so that visitors could protect themselves from contagion.<sup>1</sup>

The following brief excerpt is from Blaschke's own description<sup>2</sup> of the hospital during the time he was stationed at the capital.

---

"The men's hospital building is about nine fathoms long and eight fathoms wide. A hallway extends from the entrance, which is concealed by the long dimension, to the right of which is a room designated for the reception of patients and two wards; on the left side of the hallway is the kitchen, the pharmacy and a third ward. This corridor extends beyond the house and is joined on the left by the bath house. At the end of it are located the latrines, which are washed by seawater at high tide. In the rather spacious upper floor the medicines and the wooden chests for storing soiled linens are kept, and there is a place for hanging and drying the herbs collected in the region. Next to the kitchen is a small room for the surgeon. On either side of the entrance are small rooms—one where the clean linen is kept, the other for receiving

the jars and food containers brought on some days from the warehouse. The hallway can be heated and the wards are rather spacious and agreeable, and lighted by rather large windows. The floor is painted an olive color. The twenty-four beds are painted olive-green, but if the number of patients is increased, four more beds are added, and six patients can be placed in the reception room. The mattresses and pillows are stuffed with coconut flax, there being insufficient horsehair available. The blankets are woollen and the sheets of linen; they are changed everywhere once a week. . . . The pharmacy is rather spacious and well arranged; in a room separated from the antechamber is a furnace for distillations maintained in a sand bath and for preparing chemical compounds. Potions, plasters, and other remedies are warmed in the kitchen. . . .

Aside from the physician, the following are engaged on hospital duty in delivering care and attention to the sick: a naval surgeon of upper grade who under the physician's supervision largely performs the duties of a pharmacist; a Creole assistant qualified at the same educational level as the surgeon, and five or six Creole students who are instructed with the necessary information by the surgeon; these have the night duty on the wards, and assist the pharmacist and patients. Others include one supervisor, a cook and two helpers, and a midwife with one assistant; . . .

"The daily number of patients differs markedly according to the season, weather, and so on. There are rarely less than twenty patients in the hospital, and less than ten ambulatory patients. The number of the former, however, often grows to thirty and the latter to forty or more. All patients, including women and children, under the most favorable conditions rarely reach a total of forty in the summer, but in the fall, the winter, and sometimes even the spring, the number exceeds eighty or a hundred."

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### WARNINGS

**Liver Enzymes:** HMG-CoA reductase inhibitors, like some other lipid-lowering therapies, have been associated with biochemical abnormalities of liver function. Increases of serum transaminase (ALT, AST) values to more than 3 times the upper limit of normal occurring on 2 or more (not necessarily sequential) occasions have been reported in 1.3% of patients treated with pravastatin in the U.S. over an average period of 18 months. These abnormalities were not associated with cholestasis and did not appear to be related to treatment duration. In those patients in whom these abnormalities were believed to be related to pravastatin and who were discontinued from therapy, the transaminase levels usually fell slowly to pretreatment levels. These biochemical findings are usually asymptomatic although worldwide experience indicates that anorexia, weakness, and/or abdominal pain may also be present in rare patients.

As with other lipid-lowering agents, liver function tests should be performed during therapy with pravastatin. Serum transaminases, including ALT (SGPT), should be monitored before treatment begins, every six weeks for the first three months, every eight weeks during the remainder of the first year, and periodically thereafter (e.g., at about six-month intervals). Special attention should be given to patients who develop increased transaminase levels. Liver function tests should be repeated to confirm an elevation and subsequently monitored at more frequent intervals. If increases in AST and ALT equal or exceed three times the upper limit of normal and persist, then therapy should be discontinued. Persistence of significant aminotransferase elevations following discontinuation of therapy may warrant consideration of liver biopsy.

Active liver disease or unexplained transaminase elevations are contraindications to the use of pravastatin (see CONTRAINDICATIONS). Caution should be exercised when pravastatin is administered to patients with a history of liver disease or heavy alcohol ingestion (see CLINICAL PHARMACOLOGY: Pharmacokinetics/Metabolism). Such patients should be closely monitored, started at the lower end of the recommended dosing range, and titrated to the desired therapeutic effect.

**Skeletal Muscle: Rhabdomyolysis with renal dysfunction secondary to myoglobinuria has been reported with pravastatin and other drugs in this class.** Uncomplicated myalgia has also been reported in pravastatin-treated patients (see ADVERSE REACTIONS). Myopathy, defined as muscle aching or muscle weakness in conjunction with increases in creatine phosphokinase (CPK) values to greater than 10 times the upper limit of normal was reported to be possibly due to pravastatin in only one patient in clinical trials (<0.1%). Myopathy should be considered in any patient with diffuse myalgias, muscle tenderness or weakness, and/or marked elevation of CPK. Patients should be advised to report promptly unexplained muscle pain, tenderness or weakness, particularly if accompanied by malaise or fever. **Pravastatin therapy should be discontinued if markedly elevated CPK levels occur or myopathy is diagnosed or suspected.** Pravastatin therapy should also be temporarily withheld in any patient experiencing an acute or serious condition predisposing to the development of renal failure secondary to rhabdomyolysis, e.g., sepsis, hypotension; major surgery; trauma; severe metabolic, endocrine, or electrolyte disorders; or uncontrolled epilepsy.

The risk of myopathy during treatment with lovastatin is increased if therapy with either cyclosporine, gemfibrozil, erythromycin, or niacin is administered concurrently. There is no experience with the use of pravastatin together with cyclosporine. Myopathy has not been observed in clinical trials involving small numbers of patients who were treated with pravastatin together with niacin. One trial of limited size involving combined therapy with pravastatin and gemfibrozil showed a trend toward more frequent CPK elevations, and patient withdrawals due to musculoskeletal symptoms in the group receiving combined treatment as compared with the groups receiving placebo, gemfibrozil, or pravastatin monotherapy. Myopathy was not reported in this trial (see PRECAUTIONS: Drug Interactions). One patient developed myopathy when clofibrate was added to a previously well tolerated regimen of pravastatin; the myopathy resolved when clofibrate therapy was stopped and pravastatin treatment continued. **The use of fibrates alone may occasionally be associated with myopathy. The combined use of pravastatin and fibrates should generally be avoided.**

### PRECAUTIONS

**General:** Pravastatin may elevate creatine phosphokinase and transaminase levels (see ADVERSE REACTIONS). This should be considered in the differential diagnosis of chest pain in a patient on therapy with pravastatin.

**Homozygous Familial Hypercholesterolemia.** Pravastatin has not been evaluated in patients with rare homozygous familial hypercholesterolemia. In this group of patients, it has been reported that HMG-CoA reductase inhibitors are less effective because the patients lack functional LDL receptors.

**Renal Insufficiency.** A single 20 mg oral dose of pravastatin was administered to 24 patients with varying degrees of renal impairment (as determined by creatinine clearance). No effect was observed on the pharmacokinetics of pravastatin or its 3 $\alpha$ -hydroxy isomeric metabolite (SQ 31,906). A small increase was seen in mean AUC values and half-life (t<sub>1/2</sub>) for the inactive enzymatic ring hydroxylation metabolite (SQ 31,945). Given this small sample size, the dosage administered, and the degree of individual variability, patients with renal impairment who are receiving pravastatin should be closely monitored.

**Information for Patients:** Patients should be advised to report promptly unexplained muscle pain, tenderness or weakness, particularly if accompanied by malaise or fever.

**Drug Interactions:** Immunosuppressive Drugs, Gemfibrozil, Niacin (Nicotinic Acid), Erythromycin. See WARNINGS: Skeletal Muscle.

**Antipyrine:** Clearance by the cytochrome P450 system was unaltered by concomitant administration of pravastatin. Since pravastatin does not appear to induce hepatic drug-metabolizing enzymes, it is not expected that any significant interaction of pravastatin with other drugs (e.g., phenytoin, quinidine) metabolized by the cytochrome P450 system will occur.

**Cholestyramine/Colestipol:** Concomitant administration resulted in an approximately 40 to 50% decrease in the mean AUC of pravastatin. However, when pravastatin was administered 1 hour before or 4 hours after cholestyramine or 1 hour before colestipol and a standard meal, there was no clinically significant decrease in bioavailability or therapeutic effect. (See DOSAGE AND ADMINISTRATION: Concomitant Therapy).

**Warfarin:** In a study involving 10 healthy male subjects given pravastatin and warfarin concomitantly for 6 days, bioavailability parameters at steady state for pravastatin (parent compound) were not altered. Pravastatin did not alter the plasma protein-binding of warfarin. Concomitant dosing did increase the AUC and C<sub>max</sub> of warfarin but did not produce any changes in its anticoagulant action (i.e., no increase was seen in mean prothrombin time after 6 days of concomitant therapy). However, bleeding and extreme prolongation of prothrombin time has been reported with another drug in this class. Patients receiving warfarin-type anticoagulants should have their prothrombin times closely monitored when pravastatin is initiated or the dosage of pravastatin is changed.

**Cimetidine:** The AUC<sub>0-12h</sub> for pravastatin when given with cimetidine was not significantly different from the AUC for pravastatin when given alone. A significant difference was observed between the AUC's for pravastatin when given with cimetidine compared to when administered with antacid.

**Digoxin:** In a crossover trial involving 18 healthy male subjects given pravastatin and digoxin concurrently for 9 days, the bioavailability parameters of digoxin were not affected. The AUC of pravastatin tended to increase, but the overall bioavailability of pravastatin plus its metabolites SQ 31,906 and SQ 31,945 was not altered.

**Gemfibrozil:** In a crossover study in 20 healthy male volunteers given concomitant single doses of pravastatin and gemfibrozil, there was a significant decrease in urinary excretion and protein binding of pravastatin. In addition, there was a significant increase in AUC, C<sub>max</sub>, and T<sub>max</sub> for the pravastatin metabolite SQ 31,906. Combination therapy with pravastatin and gemfibrozil is generally not recommended.

In interaction studies with aspirin, antacids (1 hour prior to PRAVACHOL), cimetidine, nicotinic acid, or probucol, no statistically significant differences in bioavailability were seen when PRAVACHOL (pravastatin sodium) was administered.

**Other Drugs:** During clinical trials, no noticeable drug interactions were reported when PRAVACHOL was added to diuretics, antihypertensives, digitals, converting-enzyme inhibitors, calcium channel blockers, beta-blockers, or nitroglycerin.

**Endocrine Function:** HMG-CoA reductase inhibitors interfere with cholesterol synthesis and lower circulating cholesterol levels and, as such, might theoretically blunt adrenal or gonadal steroid hormone production. Results of clinical trials with pravastatin in males and post-menopausal females were consistent with regard to possible effects of the drug on basal steroid hormone levels. In a study of 21 males, the mean testosterone response to human chorionic gonadotropin was significantly reduced (p<0.004) after 16 weeks of treatment with 40 mg of pravastatin. However, the percentage of patients showing a  $\geq$ 50% rise in plasma testosterone after human chorionic gonadotropin stimulation did not change significantly after therapy in these patients. The effects of HMG-CoA reductase inhibitors on spermatogenesis and fertility have not been studied in adequate numbers of patients. The effects, if any, of pravastatin on the pituitary-gonadal axis in pre-menopausal females are unknown. Patients treated with pravastatin who display clinical evidence of endocrine dysfunction should be evaluated appropriately. Caution should also be exercised if an HMG-CoA reductase inhibitor or other agent used to lower cholesterol levels is administered to patients also receiving other drugs (e.g., ketoconazole, spironolactone, cimetidine) that may diminish the levels or activity of steroid hormones.

**CNS Toxicity:** CNS vascular lesions, characterized by perivascular hemorrhage and edema and mononuclear cell infiltration of perivascular spaces, were seen in dogs treated with pravastatin at a dose of 25 mg/kg/day, a dose that produced a plasma drug level about 50 times higher than the mean drug level in humans taking 40 mg/day. Similar CNS vascular lesions have been observed with several other drugs in this class.

A chemically similar drug in this class produced optic nerve degeneration (Wallenian degeneration of retinogeniculate fibers) in clinically normal dogs in a dose-dependent fashion starting at 60 mg/kg/day, a dose that produced mean plasma drug levels about 30 times higher than the mean drug level in humans taking the highest recommended dose (as measured by total enzyme inhibitory activity). This same drug also produced vestibulocochlear Wallenian-like degeneration and retinal ganglion cell chromatolysis in dogs treated for 14 weeks at 180 mg/kg/day, a dose which resulted in a mean plasma drug level 2 times that seen with the 60 mg/kg dose.

**Carcinogenesis, Mutagenesis, Impairment of Fertility:** In a 2-year study in rats fed pravastatin at doses of 10, 30, or 100 mg/kg body weight, there was an increased incidence of hepatocellular carcinomas in males at the highest dose (p<0.01). Although rats were given up to 125 times the human dose (HD) on a mg/kg body weight basis, their serum drug levels were only 6 to 10 times higher than those measured in humans given 40 mg pravastatin as measured by AUC.

The oral administration of 10, 30, or 100 mg/kg (producing plasma drug levels approximately 0.5 to 5.0 times human drug levels at 40 mg) of pravastatin to mice for 22 months resulted in a statistically significant increase in the incidence of malignant lymphomas in treated females when all treatment groups were pooled and compared to controls (p<0.05). The incidence was not dose-related and male mice were not affected.

A chemically similar drug in this class was administered to mice for 72 weeks at 25, 100, and 400 mg/kg body weight, which resulted in mean serum drug levels approximately 3, 15, and 33 times higher than the mean human serum drug concentration (as total inhibitory activity) after a 40 mg oral dose. Liver carcinomas were significantly increased in high-dose females and mid- and high-dose males, with a maximum incidence of 90 percent in males. The incidence of adenomas of the liver was significantly increased in mid- and high-dose females. Drug treatment also significantly increased the incidence of lung adenomas in mid- and high-dose males and females. Adenomas of the eye Harderian gland (a gland of the eye of rodents) were significantly higher in high-dose mice than in controls.

No evidence of mutagenicity was observed *in vitro*, with or without rat-liver metabolic activation, in the following studies: microbial mutagenicity, using mutant strains of *Salmonella typhimurium* or *Escherichia coli*; a forward mutation assay in L5178Y TK + / - mouse lymphoma cells; a chromosomal aberration test in hamster cells; and a gene conversion assay using *Saccharomyces cerevisiae*. In addition, there was no evidence of mutagenicity in either a dominant lethal test in mice or a micronucleus test in mice.

In a study in rats, with daily doses up to 500 mg/kg, pravastatin did not produce any adverse effects on fertility or general reproductive performance. However, in a study with another HMG-CoA reductase inhibitor, there was decreased fertility in male rats treated for 34 weeks at 25 mg/kg body weight, although this effect was not observed in a subsequent fertility study when this same dose was administered for 11 weeks (the entire cycle of spermatogenesis, including epididymal maturation). In rats treated with this same reductase inhibitor at 180 mg/kg/day, seminiferous tubule degeneration (necrosis and loss of spermatogenic epithelium) was observed. Although not seen with pravastatin, two similar drugs in this class caused drug-related testicular atrophy, decreased spermatogenesis, spermatocytic degeneration, and giant cell formation in dogs. The clinical significance of these findings is unclear.

**Pregnancy: Pregnancy Category X:** See CONTRAINDICATIONS.

Safety in pregnant women has not been established. Pravastatin was not teratogenic in rats at doses up to 1000 mg/kg daily or in rabbits at doses of up to 50 mg/kg daily. These doses resulted in 20x (rabbit) or 240x (rat) the human exposure based on surface area (mg/meter<sup>2</sup>). However, in studies with another HMG-CoA reductase inhibitor, skeletal malformations were observed in rats and mice. PRAVACHOL (pravastatin sodium) should be administered to women of child-bearing potential only when such patients are highly unlikely to conceive and have been informed of the potential hazards. If the woman becomes pregnant while taking PRAVACHOL (pravastatin sodium), it should be discontinued and the patient advised again as to the potential hazards to the fetus.

**Nursing Mothers:** A small amount of pravastatin is excreted in human breast milk. Because of the potential for serious adverse reactions in nursing infants, women taking PRAVACHOL should not nurse (see CONTRAINDICATIONS).

**Pediatric Use:** Safety and effectiveness in individuals less than 18 years old have not been established. Hence, treatment in patients less than 18 years old is not recommended at this time. (See also PRECAUTIONS: General.)

### ADVERSE REACTIONS

Pravastatin is generally well tolerated; adverse reactions have usually been mild and transient. In 4-month long placebo-controlled trials, 1.7% of pravastatin-treated patients and 1.2% of placebo-treated patients were discontinued from treatment because of adverse experiences attributed to study drug therapy; this difference was not statistically significant. In long-term studies, the most common reasons for discontinuation were asymptomatic serum transaminase increases and mild, non-specific gastrointestinal complaints. During clinical trials the overall incidence of adverse events in the elderly was not different from the incidence observed in younger patients.

**Adverse Clinical Events:** All adverse clinical events (regardless of attribution) reported in more than 2% of pravastatin-treated patients in the placebo-controlled trials are identified in the table below; also shown are the percentages of patients in whom these medical events were believed to be related or possibly related to the drug:

Body System/Event	All Events %		Events Attributed to Study Drug %	
	Pravastatin (N = 900)	Placebo (N = 411)	Pravastatin (N = 900)	Placebo (N = 411)
Cardiovascular				
Cardiac Chest Pain	4.0	3.4	0.1	0.0
Dermatologic				
Rash	4.0*	1.1	1.3	0.9
Gastrointestinal				
Nausea/Vomiting	7.3	7.1	2.9	3.4
Diarrhea	6.2	5.6	2.0	1.9
Abdominal Pain	5.4	6.0	2.0	3.9
Constipation	4.0	4.0	4.4	5.1
Flatulence	3.3	3.6	2.7	3.4
Heartburn	2.9	1.9	2.0	0.7
General				
Fatigue	3.8	3.4	1.9	1.0
Chest Pain	3.7	3.7	1.9	0.2
Influenza	2.4*	0.0	0.0	0.0
Musculoskeletal				
Localized Pain	10.0	9.0	1.4	1.5
Myalgia	2.7	1.0	0.6	0.0
Nervous System				
Headache	6.2	3.3	1.7*	0.2
Dizziness	3.3	3.3	1.0	0.5
Renal/Genitourinary				
Urinary Abnormality	2.4	2.9	0.7	1.2
Respiratory				
Common Cold	7.0	6.3	0.0	0.0
Rhinitis	4.0	4.1	0.1	0.0
Cough	2.6	1.7	0.1	0.0

\*Statistically significantly different from placebo.

The following effects have been reported with drugs in this class:

**Skeletal:** myopathy, rhabdomyolysis.

**Neurological:** dysfunction of certain cranial nerves (including alteration of taste, impairment of extra-ocular movement, facial paresis), tremor, vertigo, memory loss, paresthesia, peripheral neuropathy, peripheral nerve palsy.

**Hypersensitivity Reactions:** An apparent hypersensitivity syndrome has been reported rarely which has included one or more of the following features: anaphylaxis, angioedema, lupus erythematosus-like syndrome, polymyalgia rheumatica, vasculitis, purpura, thrombocytopenia, leukopenia, hemolytic anemia, positive ANA, ESR increase, arthritis, arthralgia, urticaria, asthenia, photosensitivity, fever, chills, flushing, malaise, dyspnea, toxic epidermal necrolysis, erythema multiforme, including Stevens-Johnson syndrome.

**Gastrointestinal:** pancreatitis, hepatitis, including chronic active hepatitis, cholestatic jaundice, fatty change in liver, and, rarely, cirrhosis, fulminant hepatic necrosis, and hepatoma, anorexia, vomiting.

**Reproductive:** gynecomastia, loss of libido, erectile dysfunction.

**Eye:** progression of cataracts (lens opacities), ophthalmoplegia.

**Laboratory Test Abnormalities:** Increases in serum transaminase (ALT, AST) values and CPK have been observed (see WARNINGS).

Transient, asymptomatic eosinophilia has been reported. Eosinophil counts usually returned to normal despite continued therapy. Anemia, thrombocytopenia, and leukopenia have been reported with other HMG-CoA reductase inhibitors.

**Concomitant Therapy:** Pravastatin has been administered concurrently with cholestyramine, colestipol, nicotinic acid, probucol and gemfibrozil. Preliminary data suggest that the addition of either probucol or gemfibrozil to therapy with lovastatin or pravastatin is not associated with greater reduction in LDL-cholesterol than that achieved with lovastatin or pravastatin alone. No adverse reactions unique to the combination or in addition to those previously reported for each drug alone have been reported. Myopathy and rhabdomyolysis (with or without acute renal failure) have been reported when another HMG-CoA reductase inhibitor was used in combination with immunosuppressive drugs, gemfibrozil, erythromycin, or lipid-lowering doses of nicotinic acid. (See WARNINGS: Skeletal Muscle and PRECAUTIONS: Drug Interactions.)

### OVERDOSAGE

There have been no reports of overdoses with pravastatin.

Should an accidental overdose occur, treat symptomatically and institute supportive measures as required.

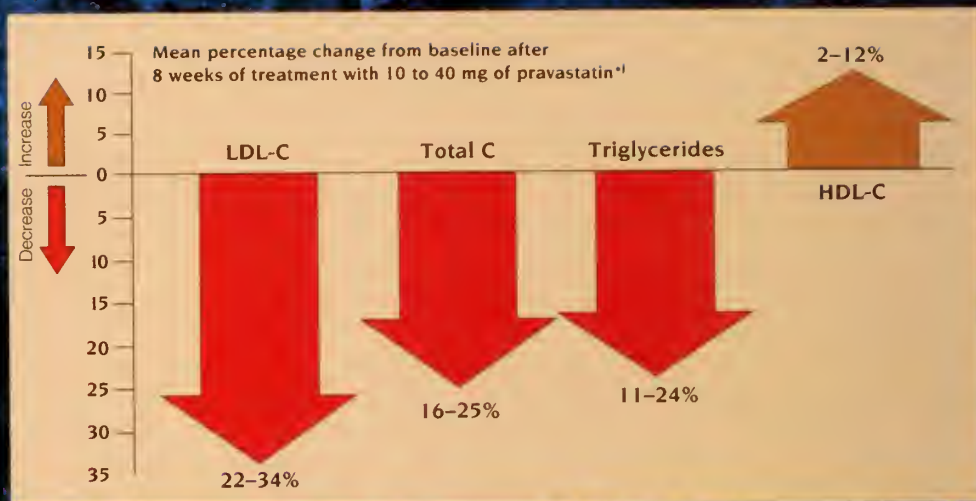




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**Reference:** 1. Jones PH, et al. Once-daily pravastatin in patients with primary hypercholesterolemia: a dose-response study. *Clin Cardiol.* 1991;14:146-151

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Please see CONTRAINDICATIONS, WARNINGS, PRECAUTIONS, and ADVERSE REACTIONS in the brief summary of prescribing information on the adjacent page.



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# ALASKA MEDICINE

Volume 35, Number 3

July/August/September 1993



*Official Journal of:*

ALASKA STATE MEDICAL ASSOCIATION  
AMERICAN SOCIETY FOR CIRCUMPOLAR HEALTH

*In this issue:* The Human Diving Response — Heat and Hypovolemia - Peripheral Vasodilators  
Vision Screening with Brückner Test by Robert W. Arnold, M.D.



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# THE ATTENUATING EFFECT OF HEAT AND HYPOVOLEMIA ON THE HUMAN DIVING RESPONSE

Robert W. Arnold, M.D.<sup>(1)</sup>

Ethan R. Nadel, Ph.D.<sup>(2)</sup>

## ABSTRACT

Seven healthy adult volunteers underwent 30-second facial immersions in ice water before and during 36 minutes exposure to intense ambient heat (68 degrees C) on separate, non-consecutive days: one normovolemic and the second with thiazide-induced, 10% hypovolemia. The normovolemic, preheat immersion induced a heart rate (H.R.) decrease from  $74 \pm 6$  to  $48 \pm 5$  bpm (-35%) and a forearm blood flow (F.B.F.) decrease from  $3.5 \pm 1.6$  to  $0.8 \pm 0.3$  ml/(100 ml min) (-77%). Pre-heat changes were similar for hypovolemic subjects. After 36 minutes of heat exposure there was a 0.7 degrees C increase in esophageal temperature and a four-fold increase in F.B.F. for normovolemic and a two-fold increase in F.B.F. for hypovolemic subjects. Heating reduced diving bradycardia more than did superimposed hypovolemia. Four subjects had diving tachycardia after hypovolemic heating. We concluded that the bradycardia response to facial immersion can be overridden by reducing the central circulating blood volume by heat and that this effect is enhanced by hypovolemia.

## INTRODUCTION

Immersion of the face in cold water elicits a profound vagal bradycardia and sympathetic peripheral vasoconstriction in many species including humans (1). The onset of diving vasoconstriction is acute and precedes the gradual onset of diving bradycardia (2). Diving vasoconstriction can be more profound than vasoconstriction due to cold pressor testing (3). The integrated response acts to maintain mean arterial pressure, and therefore to maintain cerebral perfusion during immersion (4). Facial cooling provides the afferent stimulus, mediated by the trigeminal nerve, to the human diving response. The brainstem

integration and the neural pathways of the diving response remain to be clearly delineated (5).

In order to more clearly define the sequence of events involved in the diving response, we studied the cardiovascular response to facial immersion in healthy humans exposed to conditions that would be expected to oppose the vasomotor component. Exposure to intense ambient heat causes cutaneous vasodilatation and a shift of the blood volume peripherally (6), thereby tending to reduce the filling pressure of the heart. Superimposed hypovolemia further limits the ability to maintain cardiac filling pressure and, therefore cardiac output. This was expected to jeopardize either blood pressure maintenance, or bradycardia during facial immersion. In the presence of these loads, we expect to be able to determine the hierarchy of control of the human diving response.

## METHODS

Seven healthy volunteers, five male and two female, aged 23-41, gave informed constant to participate in this study. The subjects were studied on two non-consecutive days. The first study was a control (normovolemic) experiment and the second involved rendering the subjects hypovolemic. In order to induce hypovolemic, a potassium sparing diuretic (hydrochlorothiazide 25 mg., triamterene 50 mg, p.o., b.i.d.) was administered for three days. To avoid the smooth relaxing properties of thiazides (7), no diuretics were administered on the morning of the testing. The degree of hypovolemia was assessed by comparing pre-diuretic and post-diuretic hemoglobin and hematocrit concentrations (8).

The diving response was elicited by 30 second, mid-inspiratory breath hold facial immersions ("dives") to the level of the forehead and chin in a basin of ice water at less than 3 degrees C. The maneuver was performed while the subjects were in the seated position with the legs dependent. Subjects held their breath without Valsalva

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maneuver. Heart rate (H.R.), forearm blood flow (F.B.F.), and esophageal temperature (Tes) were monitored before and during each dive. Heart rate was determined every three seconds from R-R intervals, Lead II, modified limb leads on a calibrated electrocardiograph (Hewlett-Packard 1500B). A pre-dive heart rate was obtained by averaging heart rate after 3 to 15 seconds before immersion, and the dive heart rate was obtained by averaging the heart rate from 15 to 30 seconds before of immersion. Forearm blood flow was determined 15 seconds before each dive and 15 second during the dive a using a mercury-in-silastic strain gauge and a modified Whitney venous occlusion technique (9). The esophageal temperature was continuously monitored by a thin, pliable thermocouple naso-esophageally to the level of the right atrium.

An intense heat stress was applied in a chamber illustrated in Figure 1. Two 1500W electric heaters warmed the insulated 3m x 2m x 2m enclosure from room temperature (27 degrees) to 68 degrees C. The diving response was measured at room temperature and at intervals during intense ambient heat exposure. Each subject tolerated 36 minutes of heating whether normovolemic or hypovolemic.

The degree of facial cooling during the dives was monitored by a thermocouple secured to the subject's cheek so that the temperatures of the skin-water interface could be continuously monitored. The initial dive cooled the facial skin from 32 degrees C to 10 degrees C. The facial temperature gradually rose to 36 degrees C during heat exposure, however immersion in ice water always promptly cooled the facial skin to 8-11 degrees C.

Due to the large inter-subject variability (i.e. pre-dive heart rates ranging from 50 to 85 b.p.m. before heating), heart rate and forearm blood flow data are discussed as percentage changes and are analyzed by analysis of variance (ANOVA), Global rank test (10) and paired Student-Test.

## RESULTS

**Effect of Diuretics:** Average values of weights and hemoglobin concentration before and after normovolemic heating are given in the Table. Three days of thiazide administration caused an average weight loss of 2.2 kg or 3.2% of body weight. Thiazide

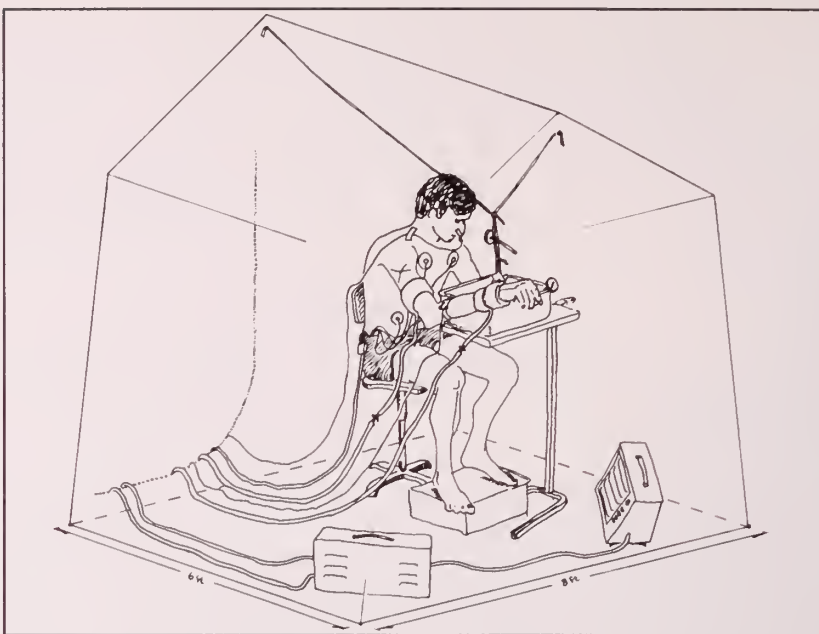


Figure 1. Diagram of a subject in the heating chamber with monitors for electrocardio-graph, venous occlusion plethysmography and esophageal temperature during apneic facial immersion in ice water.

administration also caused an increase in hemoglobin concentration from 14.2 to 16.0 gdl<sup>-1</sup>. Assuming no change in total hemoglobin, this indicated a 10.9% decrease in blood volume.

**Effects of Heat Exposure:** Exposure to 68 degree C ambient temperature for 36 minutes caused an average increase in core temperatures of 0.7 degrees C in both normovolemic and hypovolemic conditions. Immediately upon the initiation of heating, there was a transient drop in esophageal temperature lasting 10-15 minutes. Following this, esophageal temperature increased 0.04 degrees C per minute for the duration of the exposure.

**Heart Rate during Diving:** The mean and standard error heart rate changes induced by facial immersion in normovolemic and hypovolemic subjects are given in the Table. Percent changes in heart rate induced by diving before and during intense ambient heat are shown in Figure 2.

Exposure to heat significantly reduced diving bradycardia ( $F_{4,34}=21.1, p=0.0001$ ). Although all subjects consistently had diving bradycardia at room temperature, four subjects demonstrated dive heart rates 2-7 bpm higher than pre-dive levels after 36 minutes exposure to 68 degrees C. This diving *tachycardia* occurred in three subjects while hypovolemic and one subject under each blood volume state.

Comparing heat intervals, normovolemic diving bradycardia was greater than hypovolemic diving bradycardia only at 15 minutes of intense ambient heat exposure (paired t-Test = 1.96,  $p < 0.05$ ). Due to the variability

Table		NORMOVOLIC					HYPOVOLEMIC				
n=7 mean $\pm$ S.E.M		preHeat	8 min	15 min.	25 min.	36 min.	preHeat	8 min.	15 min.	25 min.	36 min.
HEART RATE (bpm)	pre-dive	74 $\pm$ 6	82 $\pm$ 2	86 $\pm$ 5	91 $\pm$ 3	98 $\pm$ 4	79 $\pm$ 7	85 $\pm$ 4	87 $\pm$ 6	98 $\pm$ 4	104 $\pm$ 4
	dive	48 $\pm$ 5	59 $\pm$ 3	68 $\pm$ 7	75 $\pm$ 5	90 $\pm$ 3	51 $\pm$ 9	71 $\pm$ 6	79 $\pm$ 6	91 $\pm$ 5	101 $\pm$ 3
BLOOD FLOW (cc/100cc min)	pre-dive	3.5 $\pm$ 1.6	3.9 $\pm$ 1.2	5.7 $\pm$ 1.6	10.32 $\pm$ 2.2	13.5 $\pm$ 1.8	2.7 $\pm$ 0.7	2.60 $\pm$ 0.7	3.6 $\pm$ 0.5	4.87 $\pm$ 0.5	5.4 $\pm$ 0.6
	dive	0.8 $\pm$ 0.3	1.87 $\pm$ 0.6	3.6 $\pm$ 1.0	7.13 $\pm$ 1.7	9.9 $\pm$ 1.8	0.8 $\pm$ 0.5	1.58 $\pm$ 0.6	2.0 $\pm$ 0.4	2.83 $\pm$ 0.5	4.2 $\pm$ 0.4
Esophageal Temperature (°C)		37.07 $\pm$ 0.17	36.95 $\pm$ 0.22	37.09 $\pm$ 0.23	37.46 $\pm$ 0.27	37.81 $\pm$ 0.27	36.89 $\pm$ 0.14	36.72 $\pm$ 0.19	36.79 $\pm$ 0.20	37.09 $\pm$ 0.21	37.40 $\pm$ 0.22
WEIGHT (Kg)		69.4 $\pm$ 4.1					67.2 $\pm$ 4.1				
HEMOGLOBIN (gm/dl)		14.2 $\pm$ 0.6					16.0 $\pm$ 0.8				

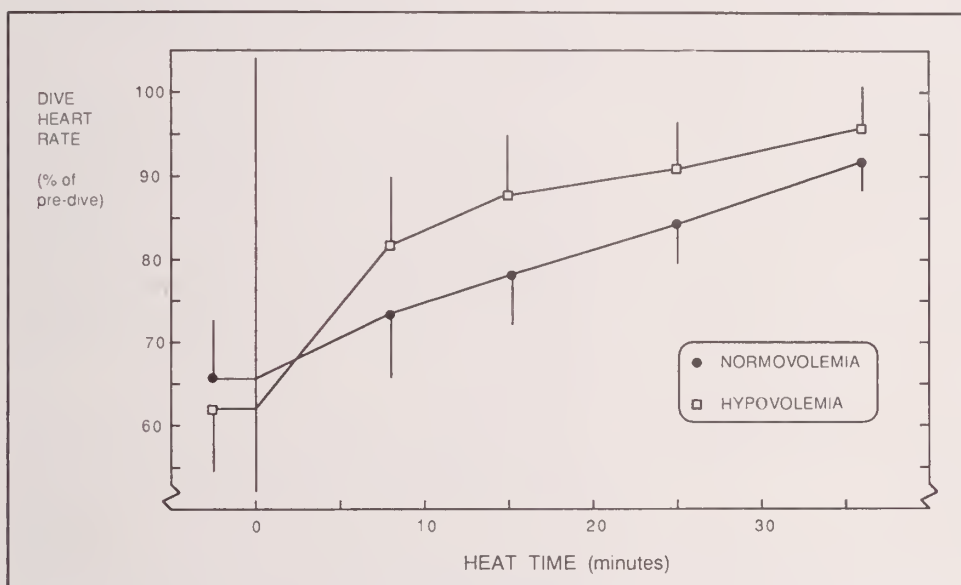


Figure 2. The effect of heat and hypovolemia on the change in heart rate induced by 30-second apneic facial immersion in ice water (mean $\pm$ SEM; n=7).

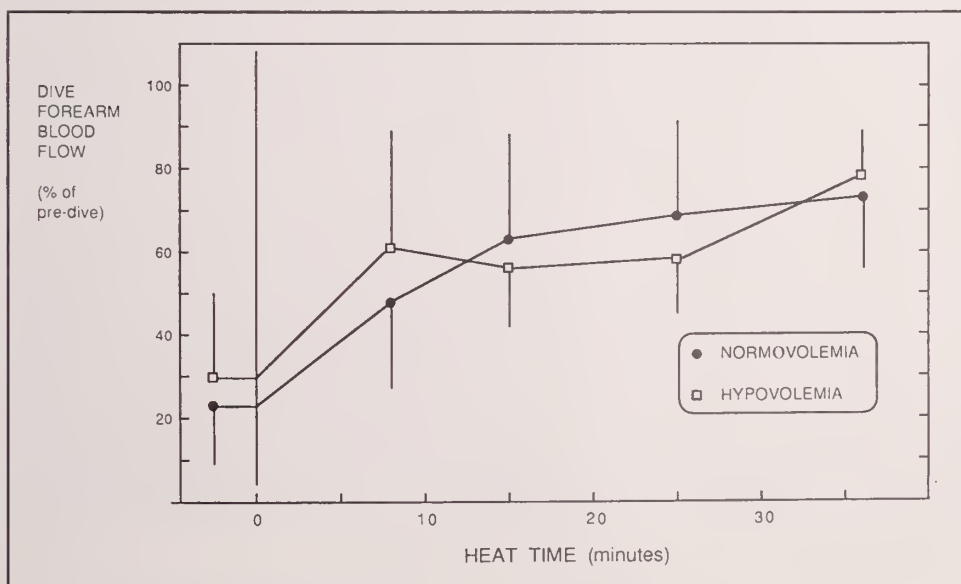


Fig 3. The effect of heat and hypovolemia on the change in forearm blood flow induced by 30-second apneic immersion in ice water (mean $\pm$ SEM; n=7).

between subjects, the effect of blood volume on diving bradycardia appeared not significant ( $F_{1,13}=5.8, p=0.12$ ), however a global ranking of bradycardia over the various heating conditions indicated a reduction in bradycardia when subjects were hypovolemic (paired t-Test =2.03,  $p=0.4$ ).

**Forearm Blood Flow during Diving:** The mean and standard error forearm blood flow changes induced by facial immersion in normovolemic and hypovolemic subjects are given in the Table. Percent changes in forearm blood flow induced by diving before and during intense ambient heat are shown in Figure 3.

Intense ambient heat induced a four-fold vasodilation when subjects were normovolemic but only a doubling of pre-dive forearm blood flow in the hypovolemic state. At room temperature, the 10.9% reduction in blood volume had no effect on the intense reduction in forearm blood flow induced by apneic facial immersion.



Diving forearm vasoconstriction was significantly reduced by exposure to intense ambient heat ( $F_{4,34}=14.9$ ,  $p=0.0001$ ). No subject had vasodilatation during facial immersion at any heat exposure interval.

Changes in blood volume had no effect on the degree of diving vasoconstriction ( $F_{1,13}=0.003$ ,  $p=.95$ ; Global paired t-Test= $1.52$ ,  $p=.44$ ).

## DISCUSSION

Prolonged exposure to sauna heating is a common ritual in certain cultures (11). Sauna temperature may be as high as 100 degrees C. After the sauna, enthusiasts generally cool off rapidly by dunking their heads in a bucket of water, by taking a cold shower or, in the extreme, by jumping through a hole cut in the ice of a frozen lake. Relatively little has been published about the cardiovascular effects of such sudden cooling. Matousek and Pribil found that the heart rate of standing subjects after sauna dropped more during a cold shower (12,13). In a study employing telemetric blood pressure data, Bachmann, et al. documented a marked, transient hypertension followed by a normotensive bradycardia accompanying whole body immersion after a 90 degrees C sauna (14). Caputa and Cabanac found a 15% bradycardia in seated, eupneic subjects during facial immersion following a 40 degrees C bath; however they failed to report pre-bath control data for comparison (15). No previous study has investigated the cardiovascular response to cold water immersion of just the face during exposure to intense ambient heat.

We observed a reduction in diving bradycardia during intense ambient heat exposure. The attenuating effect of heat was enhanced by hypovolemia. Though the vasoconstriction in the forearm during facial immersion was reduced by heat, no enhancement in this vasomotor component was attributed to superimposed hypovolemia. Venous occlusion plethysmography records changes in limb blood flow to skin and muscle vascular beds and is more representative of the arterial than the venous system (9). Recent evidence suggests that sympathetic activity during facial immersion is greater to muscle than to skin (2). Our non-invasive monitoring could not separate the relative contributions of these skin and muscle blood flow in our heated subjects. Invasive measurement of central blood pressure has given the best estimation of changes in total peripheral resistance due to selective vasoconstriction during immersion (4,14).

The attenuating effect of heat apparently was not at the surface of the face because thermocouple recordings indicated similar rates and levels of ice water immersion cooling. Since cold thermoreceptor are located very near the skin surface (16), we assume that ice water conveys a

relatively similar stimulus independent of ambient temperature.

In the present study, ambient heat produced an increase in the body temperature and a four-fold increase in resting forearm blood flow. This shift of blood flow and blood volume to the periphery resulted in attenuation of the bradycardia accompanying apneic facial immersion and inability to reduce forearm blood flow to the same extent as prior to ambient heating. Hypovolemia accelerated the reduction in diving bradycardia affected by heat in our seated subjects. Normally, the human diving response is not associated with hypotension (4). Arterial blood pressure could not be maintained during heat (vasodilatation) with the same degree of bradycardia that occurred prior to heating unless the bradycardia was accompanied by an appropriately large increase in cardiac stroke volume. This would be unlikely since stroke volume requires an adequate venous return. It appears that heat attenuates the human diving response, at least in part, by causing a peripheral redistribution of blood volume (peripheral vasodilation) that cannot be completely compensated by constriction of blood vessels in the periphery. Thus, heat and hypovolemia probably have their effects by unloading low and, if sufficiently great, high pressure baroreceptors. Blood pressure regulation (the prevention of syncope) appears to have priority over the bradycardia component of the diving response in our heated subjects.

Hypovolemia accelerated the attenuating effect of heat on the human diving bradycardia, and in some subjects, produced diving tachycardia. This supports our contention that the ability to maintain adequate cardiac filling pressure is an important variable in permitting bradycardia during diving. Diving bradycardia can be similarly reduced by alterations in posture (17) and intrapleural pressure (18), both of which compromise venous return. A decrease in cardiac preload induced by hypovolemic heat exposure would be detected by atrial stretch receptors and, if stroke volume were sufficiently lowered, by arterial baroreceptors. Stimulation of these receptors can produce a sympathetic tachycardia which may explain the diving *tachycardia* exhibited by some subjects.

Exercise and heating induce some similar physiological changes including vasodilation, an increase in heart rate, a rise in core temperature, and sweating (6,19). In contrast to the attenuating effect of heat, exercise has been associated with dramatic extremes in human diving bradycardia (20,21,22). Dynamic exercise improves venous return do to the action of the muscle pump on dependent veins. Presumably, the improved cardiac filling pressure permits a greater diving bradycardia than isometric exercise (23).

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# The Effect of Peripheral Vasodilators on the Human Diving Response

Robert W. Arnold, M.D.<sup>(1)</sup>

Ethan R. Nadel, Ph.D.<sup>(2)</sup>

## ABSTRACT

Five healthy subjects performed seated, 30-second mid-inspiratory breath hold, facial immersions in water at 1 degrees C, 10 degrees C and 25 degrees C under control conditions and under the influence of three separate vasodilators. A temperature dependent bradycardia and decrease in forearm blood flow were observed during control dives. Nitropaste produced little change in diving bradycardia. Hydralazine reduced diving bradycardia only in 1 degrees C immersion. Prazosin produced consistent reductions in temperature dependent bradycardia. There was a temperature dependent change in dive forearm blood flow which was not affected by vasodilators. This data suggest the importance of acute peripheral vasoconstriction to the bradycardia of the human diving response.

## INTRODUCTION

Profound bradycardia can be observed in conscious, seated humans during apneic facial immersion in ice water (1). This diving response involves two components of neural control on the cardiovascular system: vagal bradycardia and selective peripheral vasoconstriction. As a result, mean arterial pressure is maintained during the diving response (2).

The bradycardia of the human diving response is attenuated by mental stimulation (3), change in posture (4), and an increase in intrapleural pressure (5). Mental arithmetic causes vasodilation (6) and posture and intrapleural pressure alter venous return.

In an earlier study (7), we observed that exposure to intense ambient heat in a 68 degrees C sauna attenuated bradycardia during breath-holding and facial immersion in ice water. Eventually diving *tachycardia* resulted. We theorized that heat stress produces an attenuating effect on diving bradycardia by modifying vasomotor control during facial immersion. To test this hypothesis, we

designed the following study to measure the effect of modifications in the vasomotor component of the human diving response induced by vascular smooth muscle relaxants (hydralazine and nitroglycerine), and an agent which specifically blocks sympathetic vasoconstriction (prazosin).

## METHODS

Five healthy subjects, aged 24-42, four male and one female, gave informed consent to participate. Each was studied on four different days, one of which was a non-drug control day for the three days during which the subject was under the influence of one of three peripheral vasodilators.

The diving response was elicited by seated, 30-second, mid-inspiratory breath hold facial immersions in a basin of water. Subjects were told to avoid the Valsalva maneuver. The diving response for each subject was tested at each of three immersion water temperatures ( $1 \pm 1$  degrees C,  $10 \pm 1$  degrees C,  $25 \pm 1$  degrees C). The diving response at each water temperature was repeated thrice for each subject in a random order at three minute intervals on the control and vasodilator effected days.

Single, low dose vasodilators were administered in the following manner: one hour before one test day each subject was given one 2 mg capsule of prazosin (Minipress®, Pfizer); one hour before the next day each subject was given 50 mg of hydralazine (Apresoline®, Ciba); and 30 minutes before the last test day 15 mg of nitroglycerine paste (Nitrol® Ointment, Kremers, Urban) was applied to the skin of the shoulder of each subject.

Heart rate was obtained from R-R intervals from modified limb leads (lead II) on a continuous, calibrated electrocardiograph (Hewlett-Packard-1500B). A pre-dive heart rate was obtained from 3 to 15 seconds before the immersion and the dive heart rate was obtained by averaging heart rate from 15 to 30 seconds of immersion. Left forearm blood flow was monitored by a modified Whitney technique mercury-in-silastic strain gauge (8), venous occlusion plethysmograph 15 seconds before and 15 seconds into each 30 second immersion. Blood pressure and heart rate were determined before and after each dive.

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As with previously published human diving bradycardia experience, we observed a consistent pre-dive (0 to -5 seconds) tachycardia (9,10) and a gradual heart rate decrease for each subject occurring from 5-10 seconds after the onset of immersion. Lowest heart rates were seen between 20 and 30 seconds of immersion. Dive heart rates between 15 and 30 seconds of immersion were associated with the smallest variance over repeated trials for each subject. The degree of diving bradycardia was determined by comparing the dive average heart rate with heart rate between dives. The five subjects demonstrated a wide individual variability in both heart rate and forearm blood flow responses which is characteristic of the human diving response (9), therefore grouped average data are presented as a percentage change in heart rate and forearm blood flow rather than absolute values.

The human diving response is consistently temperature dependent such that lower immersion temperatures result in greater bradycardia (11). To compare the effect of peripheral vasodilators on the consistently temperature dependent heart rate response at all three immersion temperatures an index of the *temperature dependent diving response* (TDDR) was considered. The TDDR is the area between the interdive heart rate average and the dive heart rate average at the three immersion temperatures from 1 degrees C to 25 degrees C with the units consisting of beats per minute (b.p.m.) x degrees C.

Statistical analysis included two-way analysis of variance without replication, and the paired Student t-Test.

## RESULTS

The mean and standard error blood pressure, and the pre-dive and dive heart rates and forearm blood the

flows for control and vasodilator conditions are given in the Table. All vasodilators produced a decrease in mean arterial pressure but the decrease was significant only for nitroglycerine paste. Nitropaste produced significant decreases in both systolic ( $120 \pm 5$  to  $111 \pm 6$  mm Hg,  $p < .05$ ), and diastolic ( $73 \pm 4$  to  $61 \pm 4$  mmHg,  $p < .01$ ) pressures. Hydralazine produced no change in systolic pressure, a small decrease in diastolic pressure, but a significant ( $65 \pm 5$  to  $71 \pm 5$  bpm,  $p < .05$ ) increase in inter-dive heart rate. Prazosin at this dose produced minimal changes in inter-dive heart rate and blood pressure.

**Heart Rate:** The dive heart rate changes at three immersion temperatures under the influence of vasodilators and under control conditions are shown in Figure 1. Two-way ANOVA for relative heart rate at each immersion temperatures over the range of control and vasodilator manipulations revealed a significant variation due to individuals ( $F_{4,12} = 5.4$ ,  $p < .01$ ). Prazosin produced a significant decrease in diving bradycardia ( $F_{1,4} = 11$ ,  $p < .05$ ).

Paired t-Tests for a given immersion temperature revealed significant reductions in the degree of diving bradycardia attributable to vasodilator only for hydralazine at 1 degrees C (66% to 72%,  $p < .05$ ), and for prazosin at both 1 degrees C (66% to 83%,  $p < .05$ ) and 10 degrees C (88% to 99%,  $p < .05$ ). No significant changes in diving bradycardia were produced by nitroglycerine paste.

The *temperature dependent diving response* (TDDR) for the following conditions: control (211 bpm x degrees C), hydralazine (213 bpm x degrees C), prazosin (26 bpm x degrees C), and nitroglycerine paste (155 bpm x degrees C) revealed a significant reduction attributable only to prazosin ( $p < .05$ ).

**Forearm Blood Flow:** The dive forearm blood flow changes at three immersion temperatures under

Table

### The Effect of Peripheral Vasodilators on the Human Diving Response

N=5 mean±SEM		HEART RATE (bpm)			FOREARM BLOOD FLOW (cc/100ccxmin)			BLOOD PRESSURE	
		Immersion Temperature			Immersion Temperature			systolic (mmHg)	diastolic (mmHg)
		1°C	10°C	25°C	1°C	10°C	25°C		
CONTROL	pre-dive	69±4	64±2	65±5	3.9±0.6	3.0±0.06	3.5±0.5	120±5	73±4
	dive	39±3	52±4	54±3	1.5±0.2	1.8±0.5	2.7±0.3		
HYDRALAZINE	pre-dive	74±4	70±5	69±5	2.8±0.6	2.4±0.5	2.5±0.3	120±9	70±6
	dive	46±5*	56±4	61±6	1.3±0.2	1.5±0.4	2.0±0.4		
NITROPASTE	pre-dive	68±4	66±3	65±6	2.7±1.0	2.3±1.0	2.7±1.0	111±7*	62±4*
	dive	43±4	55±4	59±5	1.3±0.9	1.7±0.9	1.9±0.9		
PRAZOSIN	pre-dive	74±6	70±9	69±6	3.4±0.5	3.1±1.2	3.0±0.5	118±7	68±4
	dive	52±5*	62±4*	67±5	1.8±0.5	1.9±0.8	2.0±0.6		



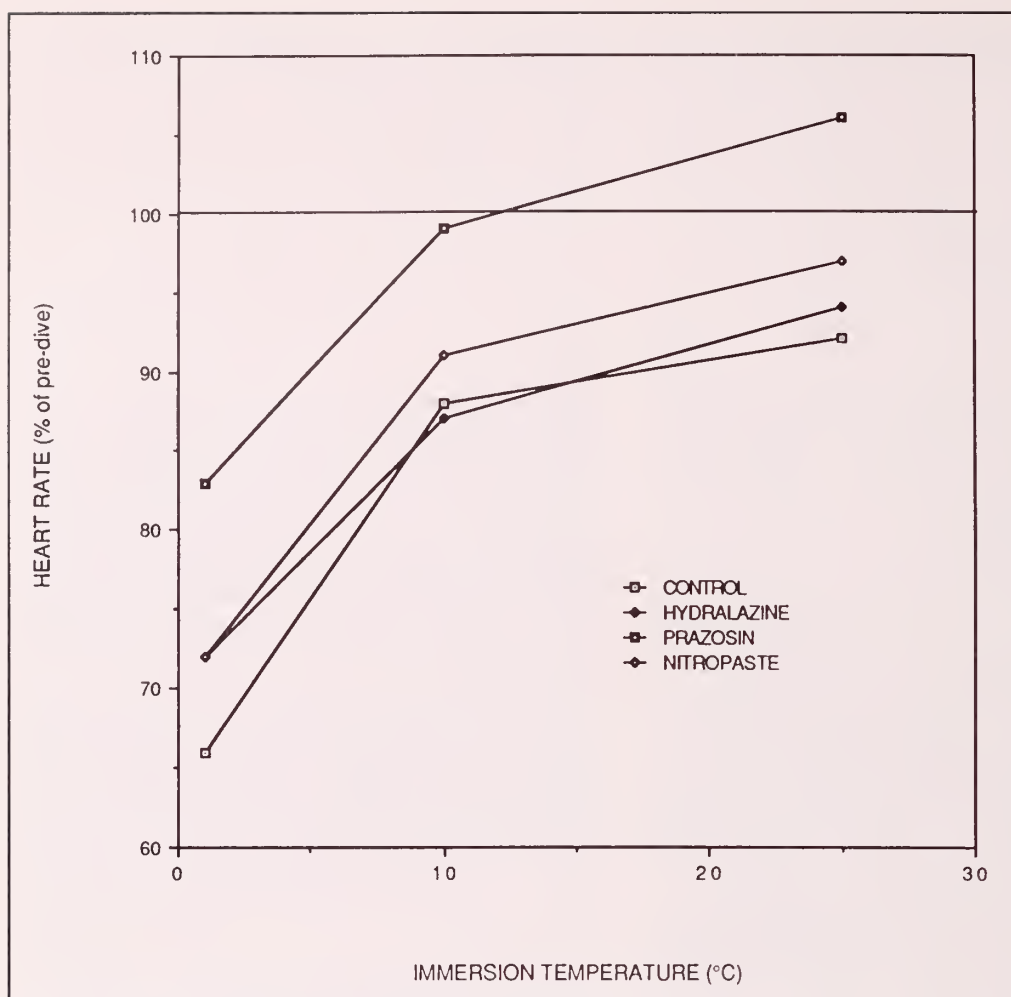


Fig. 1. The effect of low-dose peripheral vasodilators on diving bradycardia elicited by apneic facial immersion at 1 degrees C., 10 degrees C. and 25 degrees C.

influence of vasodilators and under control conditions are shown in Figure 2. The average data for five subjects reveal little change in the pre-dive forearm blood flow attributable to any of the vasodilators at these dosage levels. No significant changes in the forearm blood flow response due to any vasodilator could be found at any facial immersion temperature.

## DISCUSSION

Peripheral vasodilators which semi-specifically affect vascular smooth muscle or the neuromotor control of vasoconstriction were used by subjects during breath-hold facial immersion at ambient temperature. The doses were sufficient to produce similar, small decreases in mean arterial pressure but minimal increases in forearm blood flow. Human temperature dependent diving bradycardia was attenuated by the  $\alpha_1$ -adrenergic blocker, prazosin. A decrease in ice water diving bradycardia was also produced by the arteriolar vasodilator, hydralazine. Nitroglycerine paste caused little change in

diving bradycardia. These peripheral vasodilators had little effect on the forearm blood flow changes during the diving response; we suspect that this technique is not optimal for approximating the arterial vascular resistance.

In the only other human diving response-peripheral vasodilator trial, Finley, et. al., administered the non-specific  $\alpha$ -adrenergic agonist, phentolamine, to human subjects and investigated its effect on heart rate (12). The heart rate decreases during very brief (8 second) apneic ice water facial immersions was only slightly smaller for phentolamine than for control.

Nitroglycerine has long been used

as a vasodilator especially for the dilation of coronary vessels in angina pectoris (13,10). In low dose, this nitrite produces dilation of veins which predominates over arterioles. Nitrites reduce pre-load more than afterload, and affect a decrease in cardiac output in normal subjects. Nitroglycerine causes relatively little change in resting heart rate. The half life of nitroglycerine is 1-3 minutes and therefore it can be administered as a paste continuously absorbed through the skin. The minimum suggested dose of nitropaste (Nitrol® ointment, Kremers-Urban Co.) is 5-10 mg transdermally.

Hydralazine (Apresoline®, Ciba) is an oral or intravenous vasodilator which preferentially dilates arterioles (10,14). It nonspecifically suppresses the vascular response to pressor agents. Hydralazine affects an increase in pulmonary arterial diastolic pressure, venous return, blood flow to splanchnic, cerebral and renal vascular beds, and increases plasma renin concentration. Heart rate, stroke volume and cardiac output are elevated during hydralazine use by way of a secondary baroreflex. Hydralazine reduces diastolic more than systolic pres-

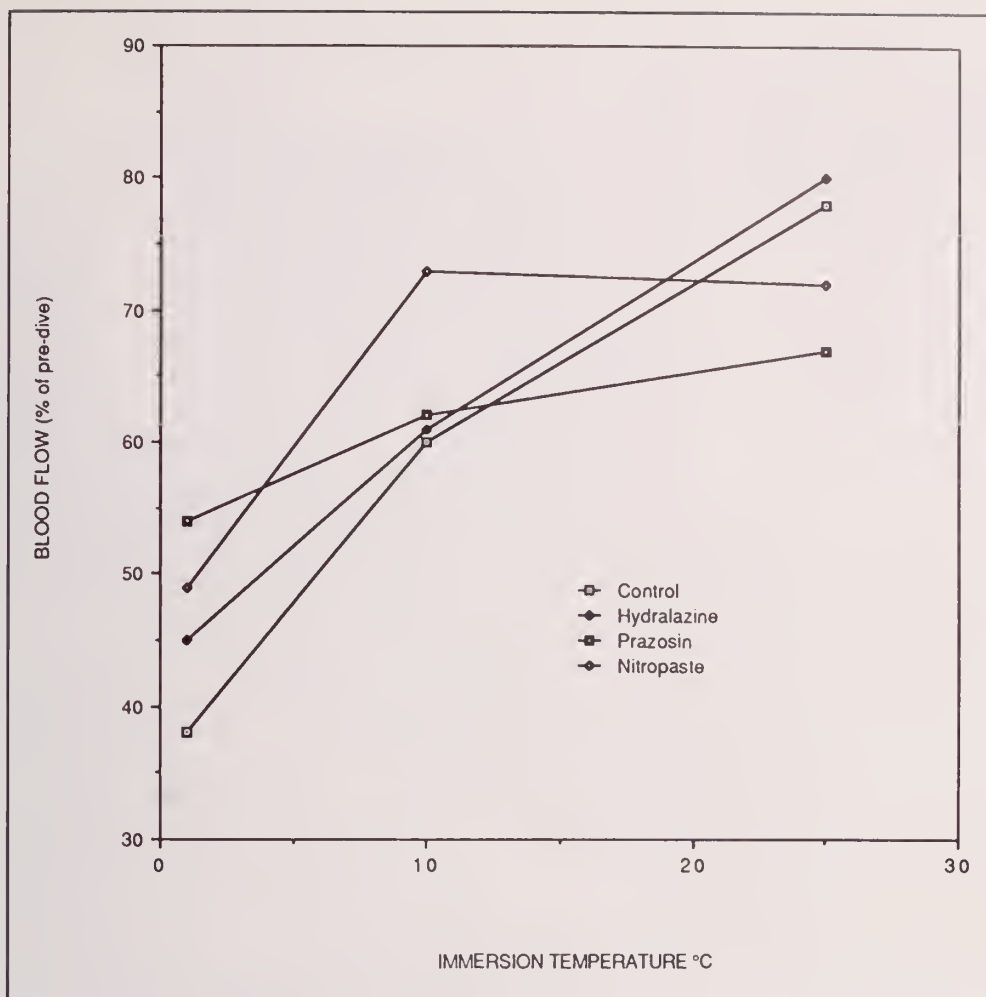


Fig. 2. The effect of low-dose peripheral vasodilators on the forearm vasomotor component of the human diving response elicited by apneic facial immersion at 1 degrees C, 10 degrees C and 25 degrees C.

sure. The half life of hydralazine is approximately 3 hours and the suggested starting minimal dose is 25 mg.

Prazosin (Minipress®, Pfizer) is a selective, post-synaptic  $\alpha_1$ -adrenergic blocking agent (15,16). It is a balanced arteriolar and venous dilator. Prazosin usage is not associated with a reflex tachycardia nor with an increase in plasma renin concentration. In patients with congestive heart failure, prazosin reduces right and left heart filling pressures, and increases stroke volume. The half life of prazosin is about 3 hours. The suggested minimum dose of prazosin is 1 mg.

Central arterial pressure, the product of cardiac output and peripheral vascular resistance, must be maintained to avoid syncope during bradycardia. Cardiac output, a product of heart rate and stroke volume, falls rapidly during diving response because stroke volume cannot completely compensate. Since prolonged extremes in human diving bradycardia are not associated with syncope, a simultaneous rapid increase in peripheral resistance must be associated with apneic facial immersion (1). We suspect that the reduced diving bradycardia

under the influence of peripheral vasodilators is a compensation for alteration in peripheral vascular resistance or venous return.

Nitrites and hydralazine affect steady reductions in vascular smooth muscle tone and therefore a gradual, eventually static decrease in peripheral resistance. Prazosin, however, inhibits acute adrenergic vasoconstriction in addition to affecting a static reduction in neurogenic vasomotor tone. The fact that prazosin, more than hydralazine and nitropaste, reduced apneic, facial immersion bradycardia may merely be dose dependent, or it may imply that the capacity for acute sympathetic vasoconstriction is a prerequisite for the human diving re-

sponse. Diving bradycardia occurs gradually over the first 15 seconds of apneic facial immersion (1) allowing central blood pressure to be maintained (2). If, during the onset of the diving response, an intense increase in peripheral resistance did not occur and blood pressure started to drop, baroreceptors and/or vascular stretch receptors could respond with tachycardia or reduced bradycardia.

Venous occlusion plethysmography measures changes in muscle and skin blood flow in a limb and is more representative of the arterial than the venous system. Baseline and relative change forearm blood flow were not greatly affected by the low-dose peripheral vasodilators in this study. Under control conditions, a relatively linear relationship exists between diving heart rate changes and changes in forearm blood flow (17,10). Blood flow is reduced more in the calf than the forearm during immersion (18) and finger blood flow reduction precedes the decrease in the forearm (19). Sympathetic nerve fascicle activity to skeletal muscle precedes diving bradycardia while sympathetic activity to the skin of the



toe was inhibited (20). Peripheral vasoconstriction in the diving response affects some vascular beds more than others. Venous occlusion plethysmography of a limb grossly approximates vasomotor changes under many conditions but is inferior to continuous monitoring of arterial pressure (2) when measuring the total peripheral vascular resistance during the human diving response.

## ACKNOWLEDGMENT

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THE COMMITMENT CONTINUES



# American Society for Circumpolar Health

## Message from the President

First, let me thank the members of the Society for electing me President for the next three years. It is of course a great honor, but it is also a challenge to me to make the Society a truly positive force in the quest for improved health in the north.

I will take this opportunity to give a broad overview of what I think the general direction the Society should take. This will be followed by some announcements and a specific request to membership.

Considering that I received 77 percent of the vote, it is my assumption that the members look for leadership with some emphasis on education and medical research, my areas of expertise. I will therefore, with the help of the membership, chart a course for the Society that will stress prevention of health problems through education and research. Such a course will ultimately lead to better health status and prospects for all people living at high latitudes. This can be achieved by all of us working together, if we improve communication between those concerned with health issues and set simple, attainable goals. Let's do the simple and most relevant things first and then build on those achievements for attaining more difficult goals.

It is not responsible to say that the health status and prospects for good health are acceptable for people living in the north. There is a lot to be done in many areas. In the area of health education, Alaska is certainly failing. The University of Alaska, for example, fails to recognize that health is everyone's first priority and the most important business in the State. There is no policy on Health Sciences at the University. The result is that our students get basically no education about such important issues as nutrition and communicable diseases. A campus may offer 40 courses in pottery making and painting and not a single course that explains how AIDS and hepatitis A are transmitted. The problem is compounded when some of our graduates become teachers, not being able to answer the simplest questions related to health. Surely the universities should be given the resources to correct this important error in priorities. The Society of Circumpolar Health can have a very positive effect by each member writing their state legislators and the university regents about this issue. I will shortly appoint a committee to evaluate health education in our schools and universities in order to enable the Society for Circumpolar Health to offer specific recommendations.


Much is also to be done in the area of relevant biomedical research. I do not need to tell you that we have a number of serious health problems in the north not understood by science and health professionals. These include, but are not limited to, seasonal depression (which affects some 25 percent of the population in Alaska), and alcoholism (FAS affects as many as one out of four infants in our Eskimo population). When Nixon closed the Arctic Health Research Center in Fairbanks in 1972, arctic medical research was dealt a blow from which it has never recovered. The abandonment of vital research priorities by Washington led to such a decrease in medical research activities in Alaska, that when I came to Fairbanks in 1985, only one NIH grant was awarded in Alaska. This has since increased to about ten out of some 20,000 awarded each year by NIH. Alaska receives about \$800,000 a year out of \$6 billion awarded each year. Thus, Alaska receives \$1.50 per capita per year in NIH research funds while the average state receives some \$30.00. NIH also funds some 900 research centers throughout the U.S., but not a single one in Alaska. Simply put, Arctic Medical Research is not receiving its fair share of NIH research funds. As a society, we must work on remedying this situation. There will be more on this later.

These thoughts are among the few that occur to me as we begin three challenging and exciting years together.

The IX International Congress on Circumpolar Health in Iceland was a great success. Our hosts were wonderful and we were all pleased to see the high quality of the research reports. The Board of the Society meets this week to plan strategy for the X International Congress on Circumpolar Health in 1996 and other activities of the Society. More about this later, suffice it to be said here that John Middaugh has agreed to serve as the President of Congress and that he is already making good progress in laying the groundwork and raising money.

Over the next three years I will ask you to participate in specific actions that will hopefully contribute to solving our health problems. In this first message to the Society, I would also like to suggest that we all sign up at least one new member of the Society. In order for us to be successful in achieving any goal we need more participants and a broader base of support.

Sven O. E. Ebbesson, Ph.D.  
President,  
American Society for Circumpolar Health



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# Vision Screening in Alaska: Experience with Enhanced Brückner Test

Robert W. Arnold, M.D.

## INTRODUCTION

Most Alaskan children see their scenic state well. However, a few children have vision threatening conditions. It is my goal to detect and treat all such young Alaskans as soon, and as thoroughly, as possible.

Children are not easily screened for visual disorders. They are often not able to verbalize symptoms. They frequently don't sit still. They object to close bright lights shining in their eyes. Some verbal children with monocular visual disabilities have learned to cheat on screening eye exams yielding false negative results. Irreversible amblyopia can occur if imperfect vision is experienced during the first ten years of life.

I recommend a quick, inexpensive, non-threatening and non-verbal technique to enhance the visual screening of Alaskan children. This test is called Enhanced Brückner Test.

## METHODS

The Enhanced Brückner Test should be used *in addition to* the distance and near chart verbal tests which currently are administered by school nurses, optometrists, physicians and community health screens. Children who cannot be annually examined by a primary care physician should have an eye examination at age one year. All Alaskan children should have a thorough eye exam including cycloplegic refraction and an assessment of the retina by age five years.

The vision in non-verbal children is assessed by fixation responses. Each normal unoccluded eye should be able to center on an interesting object without nystagmus. When the occluder is removed, the normal eye should maintain fixation. Children object to occlusion of a monocularly poorly seeing eye, but do not object to occlusion of a blind eye. Normal vision in infants can be reported as **SCM, ou** or "Steady, Centered and Maintained, Each Eye."

The Brückner Test requires repeated sighting through a direct ophthalmoscope from a distance of one-half meter in reduced ambient light (Figure 1). The objective



Fig 1. The Enhanced Brückner Test: View each pupil with a direct ophthalmoscope from one-half meter.

lens should be plano (zero). The coaxial illumination of the direct ophthalmoscope will yield a red pupillary reflex to be simultaneously compared in both eyes. The Brückner Test is positive if red reflex asymmetry persists. Positive Brückner Tests are highly sensitive and fairly specific for large and small angle strabismus, obstructions in the ocular media, refractive errors and retinal disorders (1). In large or small angle strabismus, the fixing eye has a darker red reflex (2). Mydriatics (i.e. tropicamide-Mydracyl®) can be used if the pupils fail to dilate sufficiently.

The Brückner Test can be enhanced to detect asymmetric neuroretinal dysfunction and additional amblyogenic refractive errors. After initial simultaneous bilateral viewing, the ophthalmoscope beam is directed from one eye to the other several times. A relative afferent pupillary defect or Marcus-Gunn will appear as more pupil constriction with the light in the better eye and relative dilation as the light swings to the impaired eye (3). The fixed viewing distance obviates unwanted accommodative pupillary constriction. Retinal detachment, optic nerve hypoplasia, optic nerve tumor or atrophy can be detected by asymmetric Enhanced Brückner pupils. Amblyogenic refractive errors can be detected by viewing just over the top and the side of the direct ophthalmoscope aimed at relatively large pupils. High or asymmetric myopia, hyperopia and astigmatism can be detected as a non-uniform red reflex crescent.

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Table <b>Childhood Eye Disease and Examination Techniques:</b>							
EBT = Enhanced Brückner Test EXT = External Observation SL = Split Lamp Fun = fundus by direct or indirect ophthalmoscopy Ret = Retinoscopy Tono = Tonometry, i.e. Tonopen® VA = Snellen or Allen distance acuity							
DISORDER	EBT	EXT	SL	Fun	Ret	Tono	VA
Vision Threatening							
Amblyopia:							
Strabismic-Large Angle	**	*					*
Strabismic-small angle	**						*
Deprivational (cataract)	**		*		*		*
Refractive	*				**		*
Keratitis	*	*	**		*		*
Iritis	*		**		*		
Glaucoma		*	*	**		*	
Retinal Detachment	*			**			
Shaken Baby	*			**			
Neuroretinal Dysfunction	*			*			*
Life Threatening							
Retinoblastoma	*			**			*
Orbital Tumor		*					
Orbital Cellulitis		*					
Optic Nerve Glioma	*			*			*
Brain Tumor/papilledema				*			

An additional enhancement of the Brückner Test extends the attentive fixation of wiggly, small children. The direct ophthalmoscope can be aimed over a small toy and the beam can be alternated from white to green (red-free) which may give a few seconds more to compare red reflexes.

## OBSERVATIONS

Various ophthalmic tests beneficial for screening vision and life threatening eye disease in children are compared in the Table. Notice how many can be seen using simple external observation and the Enhanced Brückner Test.

Figures 2-15 show photographs of positive Enhanced Brückner Tests (Olympus OM-2S with T-32 Flash, Tokina 500mm Mirror f.8, Ektachrome 100 ASA, distance 3 meters). Each Alaskan child was referred as a result of positive conventional or Brückner Test Screening.

## CONCLUSION

With practice, the Enhanced Brückner Test takes less than ten seconds to administer. In children, more ocular disease can be detected than with direct ophthalmoscopy through undilated pupils. Photoscreening devices are available commercially (4). Each uses a camera or video with a relatively coaxial flash and a fixed-distance chin rest. Photographs from these usually give reliable infor-

mation on most children whose pupils dilate sufficiently in a dim room. Primary physicians and public health nurses may extend detection of visual disorders with these devices. However, the photographs are less reliable and slightly more expensive than the Enhanced Brückner Test when used by an experienced observer. I urge any care-giver charged with the awesome responsibility of screening young Alaskans for visual disorders to practice the Enhanced Brückner Test. Please refer young Alaskans with persistently positive tests to an appropriate ophthalmologist or optometrist for confirmation.

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Fig 2. Asymmetric Red Reflex (Positive Brückner) due to small angle (4 pd) esotropia (microtropia or monofixation syndrome).



Fig 3. Positive Brückner due to small angle (15 pd) esotropia. Darker red reflex in patient's left fixing eye.



Fig 4. Congenital Glaucoma, right eye. Notice the asymmetry of corneal size.



Fig 5. Bilateral ectopia lentis (dislocated lenses) which may be due to Marfan's or Homocystinuria.



Fig 6. Left congenital cataract.



Fig 7. Bilateral keratoconus yields "oil drop" appearance in red reflex.



Fig 8. Vertically viewed Enhanced Brückner of high astigmatism right eye.



Fig 9. Horizontally viewed Enhanced Brückner of high astigmatism right eye, same patient as Fig 8.

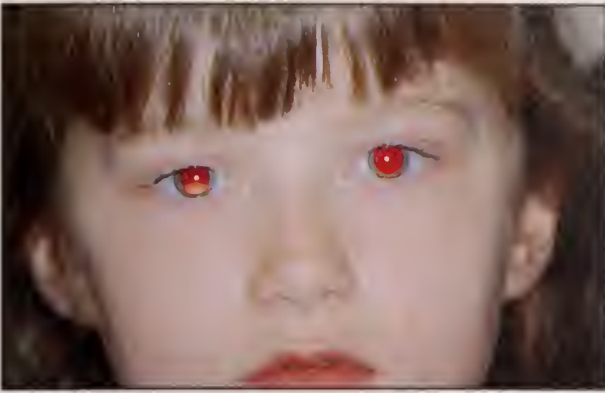


Fig 10. Amblyogenic anisometric hyperopia (+6.50 od, +1.00 os).



Fig 11. Anisometropia. Same patient as Figure 10 treated with a contact lens.



Fig 12. Yellow reflex due to Coat's Disease, a potentially blinding childhood retinal vascular disorder in the left eye.



Fig 13. Large herpes dendritic lesion on the left cornea.



Fig 14. Posterior synechia due to iritis in a patient with Juvenile Rheumatoid Arthritis.



Fig 15. Bilateral Retinoblastoma causing leukocoria.



# The Effect of Granulocyte-Macrophage Colony Stimulating Factor on Paroxysmal Nocturnal Hemoglobinuria:

## A Case Report

by Dale I. Webb, M.D.<sup>(1)</sup>  
Joan L. Bundtzen, M.D.<sup>(2)</sup>

### ABSTRACT

This case report concerns a woman thought to have a low blood count myelodysplastic syndrome who responded poorly to cytosine arabinoside. She was treated with Granulocyte-Macrophage Colony Stimulating Factor (GM-CSF) and had an adverse reaction resembling a capillary leak syndrome. Later on, an episode of post-transfusion hemoglobinuria led to the correct diagnosis of paroxysmal nocturnal hemoglobinuria (PNH). This case report suggests an adverse effect of GM-CSF on PNH.

### TEXT

A 69-year old Caucasian lady presented at the age of 62 with pancytopenia, (see table). She had no history of

thrombotic episodes, anemia, or urine discoloration. Her initial reticulocyte count was 6.7 percent but on repeat, 1.7 percent. LDH values were elevated with an increase in fraction one. Total bilirubin ranged from 1.0 - 1.6. Renal function and liver function tests were normal. The bone marrow initially was hypocellular with some erythroid hyperplasia. Subsequent marrows were normocellular to hypocellular. Cytogenetic studies were initially normal but later showed deletion of the short arm of chromosome #12 in about a third of the cells. She was diagnosed as having myelodysplastic syndrome.

Over the first 54 months of the patient's course transfusions were given at a rate of about three units every three months when the hematocrit dropped into the low 20s and she complained of head "pounding." Platelets were given when platelet counts fell below 20 thousand and there were signs of bleeding. She required no transfusion over the next 24 months.

During month 30 a venous access device was placed and thereafter she was given coumadin 1 or 2 mg. per day.

Treatment with vitamins B12 and folic acid was given the first several months during which time the patient underwent an uneventful abdominal panhyserec-tomy. In month 21 Ara-C was given 10 mg/M<sup>2</sup> every 12 hours for 21 days. Ensuing low blood counts led to the cessation of Ara-C. During the 31st month GM-CSF was given subcutaneously in escalating doses from 60 µg/M<sup>2</sup>/day to 480 µg/M<sup>2</sup>/day over 28 days, (divided doses twelve hours apart).

For 28 days the patient tolerated GM-CSF with only mild fevers and vague abdominal discomfort. However, in the five day period after stopping GM-CSF she rapidly developed fever, abdominal and mu-

**Table**

#### Clinical Course

(Lowest counts chosen neareach 6 month or treatment interval.)

Month	Hematocrit	WBC	Platelet	Treatment
0	27%	1.7k	71k	Vit B12 & Folic a.
12	24%	1.9k	61k	Ara-C
	22%	0.7k	11k	
24	18%	1.3k	43k	GM-CSF
	31%	8.0k	1k	
36	21%	1.8k	73k	Prednisone
	23%	2.1k	82k	
48	26%	2.3k	11k	
	30%	2.4k	81k	
60	32%	4.6k	127k	"
	29%	5.7k	147k	"

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cous membrane pain, swelling of the abdomen, lower extremity edema and multiple bruises. Imaging studies showed hepato-splenomegaly and ascites. The hematocrit and WBC increased but the platelet count fell to one thousand. Differential white cell count showed 23 percent bands and 20 percent eosinophils. A bone marrow study revealed an increased number of megakaryocytes. The PTT was 33.3, PT 13.1, albumin was 2.6, LDH 617, total bilirubin 1.7, SGOT 28, SGPT 68, and Fibrinogen 275. Fibrin Split Products were not found. The patient responded to transfusions, gamma globulin intravenously and corticosteroids. By month 24 hemogram values returned to pretreatment levels.

At month 54 the patient and her nurse reported dark urine after red cell transfusion. Sugar water and Ham's tests were positive, the latter showed 37.5 percent type III cells and 63.5 percent type II (performed by Sharon Hoffman in the Lane Research Lab, Wendell F. Rosse, M.D., Director, Duke University Medical Center, Durham, N.C.).

## DISCUSSION

The sugar water test is used more for screening than the more definitive Ham test. Both tests detect lysis of the complement sensitive PNH red cells. By using different amounts of complement, the PNH red cell can be classified as to their sensitivity to lysis. In this patient 37.5 percent of her cells exhibited the highest degree of sensitivity.

PNH is an acquired disease of the bone elements characterized by hypoplasia of the marrow, hemolytic anemia, thrombotic episodes and a propensity to end in leukemia. The hemolysis, a hallmark of this disease, is caused by activated complement. The normal red cell membrane contains glycosyl phosphatidyl inositol which holds, on the cell surface, at least nine proteins, some of which (for instance CD 55 and 59) are crucial in blocking the lytic attack of complement. Apparently a defect in that anchoring system causes loss of those protective proteins, which then leads to hemolysis (1). The abnormal biochemistry, however, fails to explain thromboses and leukemia (2). Nor do these abnormalities explain in this patient the observed effects of GM-CSF which seemed not to be accelerated hemolysis, but rather thrombocytopenia, left shifted neutrophils and eosinophilia. The patient's clinical response suggested a capillary leak syndrome (3).

The "capillary leak syndrome," characterized by edema and effusions, is a well described toxic effect of GM-CSF, along with the more usual problems of pain, rashes, fever and thrombosis. GM-CSF causes an increase in monocytes, neutrophils and eosinophils. As in this patient, who developed an eosinophile count of 1,600, the eosinophilia can be intense (4). Specula-

tion has it that this kind of cell elevation produces the blood vessel leak. The peculiarity in this patient was that the reaction to GM-CSF was so severe, and thrombo-cytopenia so life threatening.

Often patients with PNH, as with this case, experience a worsening of their hemolysis after blood transfusions. The usual explanation for this is that complement in the transfusion accelerates the hemolysis. Thus washed red cells are frequently recommended in PNH patients. This practice has been questioned in favor of filtered, group specific blood (5).

## CONCLUSIONS

PNH, which may masquerade as aplastic anemia and myelodysplasia, may be made worse by GM-CSF.

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(continued on page 224)

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**VASERETIC<sup>®</sup> 10-25**  
Enalapril Maleate-Hydrochlorothiazide

*Next*

Dosage must be individualized; the fixed combination is not for initial therapy.

Evaluation of the hypertensive patient should always include assessment of renal function.

For a Brief Summary of Prescribing Information, see adjacent pages.



**TABLETS**  
**VASERETIC®**  
(ENALAPRIL MALEATE-HYDROCHLOROTHIAZIDE)

**USE IN PREGNANCY:** When used in pregnancy during the second and third trimesters, ACE inhibitors can cause injury and even death to the developing fetus. When pregnancy is detected, VASERETIC® (Enalapril Maleate-Hydrochlorothiazide) should be discontinued as soon as possible. See WARNINGS, Fetal/Neonatal Morbidity and Mortality.

**CONTRAINDICATIONS:** VASERETIC is contraindicated in patients who are hypersensitive to any component of this product and in patients with a history of angioedema related to previous treatment with an angiotensin converting enzyme inhibitor. Because of the hydrochlorothiazide component, this product is contraindicated in patients with anuria or hypersensitivity to other sulfonamide-derived drugs.

**WARNINGS:** General: Enalapril Maleate; Hypotension: Excessive hypotension was rarely seen in uncomplicated hypertensive patients but is a possible consequence of enalapril use in severely salt/volume depleted persons such as those treated vigorously with diuretics or patients on dialysis.

Syncope has been reported in 1.3 percent of patients receiving VASERETIC. In patients receiving enalapril alone, the incidence of syncope is 0.5 percent. The overall incidence of syncope may be reduced by proper titration of the individual components. (See PRECAUTIONS, Drug Interactions, and ADVERSE REACTIONS.)

In patients with severe congestive heart failure, with or without associated renal insufficiency, excessive hypotension has been observed and may be associated with oliguria and/or progressive azotemia, and rarely with acute renal failure and/or death. Because of the potential fall in blood pressure in these patients, therapy should be started under very close medical supervision. Such patients should be followed closely for the first two weeks of treatment and whenever the dose of enalapril and/or diuretic is increased. Similar considerations may apply to patients with ischemic heart or cerebrovascular disease, in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident.

If hypotension occurs, the patient should be placed in the supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses, which usually can be given without difficulty once the blood pressure has increased after volume expansion.

**Angioedema:** Angioedema of the face, extremities, lips, tongue, glottis and/or larynx has been reported in patients treated with angiotensin converting enzyme inhibitors, including enalapril. In such cases VASERETIC should be promptly discontinued and appropriate therapy and monitoring should be provided until complete and sustained resolution of signs and symptoms has occurred. In instances where swelling has been confined to the face and lips the condition has generally resolved without treatment, although antihistamines have been useful in relieving symptoms. Angioedema associated with laryngeal edema may be fatal. Where there is involvement of the tongue, glottis or larynx, likely to cause airway obstruction, appropriate therapy, e.g., subcutaneous epinephrine solution 1:1000 (0.3 mL to 0.5 mL) and/or measures necessary to ensure a patent airway, should be promptly provided. (See ADVERSE REACTIONS.)

Patients with a history of angioedema unrelated to ACE inhibitor therapy may be at increased risk of angioedema while receiving an ACE inhibitor (see also CONTRAINDICATIONS).

**Neutropenia/Agranulocytosis:** Another angiotensin converting enzyme inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment especially if they also have a collagen vascular disease. Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates. Marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded. Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered.

**Hydrochlorothiazide:** Thiazides should be used with caution in severe renal disease. In patients with renal disease, thiazides may precipitate azotemia. Cumulative effects of the drug may develop in patients with impaired renal function.

Thiazides should be used with caution in patients with impaired hepatic function or progressive liver disease, since minor alterations of fluid and electrolyte balance may precipitate hepatic coma.

Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma.

The possibility of exacerbation or activation of systemic lupus erythematosus has been reported.

Lithium generally should not be given with thiazides (See PRECAUTIONS, Drug Interactions, Enalapril Maleate and Hydrochlorothiazide).

**Pregnancy, Enalapril-Hydrochlorothiazide:** There was no teratogenicity in rats given up to 90 mg/kg/day of enalapril (150 times the maximum human dose) in combination with 10 mg/kg/day of hydrochlorothiazide (2 1/2 times the maximum human dose) or in mice given up to 30 mg/kg/day of enalapril (50 times the maximum human dose) in combination with 10 mg/kg/day of hydrochlorothiazide (2 1/2 times the maximum human dose). At these doses, fetotoxicity expressed as a decrease in average fetal weight occurred in both species. No fetotoxicity occurred at lower doses; 30/10 mg/kg/day of enalapril-hydrochlorothiazide in rats and 10/10 mg/kg/day of enalapril-hydrochlorothiazide in mice.

When used in pregnancy during the second and third trimesters, ACE inhibitors can cause injury and even death to the developing fetus. When pregnancy is detected, VASERETIC should be discontinued as soon as possible. (See Enalapril Maleate, Fetal/Neonatal Morbidity and Mortality, below.) Enalapril Maleate, Fetal/Neonatal Morbidity and Mortality: ACE inhibitors can cause fetal and neonatal morbidity and death when administered to pregnant women. Several dozen cases have been reported in the world literature. When pregnancy is detected, ACE inhibitors should be discontinued as soon as possible.

The use of ACE inhibitors during the second and third trimesters of pregnancy has been associated with fetal and neonatal injury, including hypotension, neonatal skull hypoplasia, anuria, reversible or irreversible renal failure, and death. Oligohydramnios has also been reported, presumably resulting from decreased fetal renal function; oligohydramnios in this setting has been associated with fetal limb contractures, craniofacial deformation, and hypoplastic lung development. Prematurity, intrauterine growth retardation, and patent ductus arteriosus have also been reported, although it is not clear whether these occurrences were due to the ACE-inhibitor exposure.

These adverse effects do not appear to have resulted from intrauterine ACE-inhibitor exposure that has been limited to the first trimester. Mothers whose embryos and fetuses are exposed to ACE inhibitors only during the first trimester should be so informed. Nonetheless, when patients become pregnant, physicians should make every effort to discontinue the use of VASERETIC as soon as possible.

Rarely (probably less often than once in every thousand pregnancies), no

10  
mg

25  
mg

alternative to ACE inhibitors will be found. In these rare cases, the mothers should be apprised of the potential hazards to their fetuses, and serial ultrasound examinations should be performed to assess the intraamniotic environment.

If oligohydramnios is observed, VASERETIC should be discontinued unless it is considered lifesaving for the mother. Contraction stress testing (CST), a non-stress test (NST), or biophysical profiling (BPP) may be appropriate, depending upon the week of pregnancy. Patients and physicians should be aware, however, that oligohydramnios may not appear until after the fetus has sustained irreversible injury.

Infants with histories of *in utero* exposure to ACE inhibitors should be closely observed for hypotension, oliguria, and hyperkalemia. If oliguria occurs, attention should be directed toward support of blood pressure and renal perfusion. Exchange transfusion or dialysis may be required as means of reversing hypotension and/or substituting for disordered renal function. Enalapril, which crosses the placenta, has been removed from neonatal circulation by peritoneal dialysis with some clinical benefit, and theoretically may be removed by exchange transfusion, although there is no experience with the latter procedure.

No teratogenic effects of enalapril were seen in studies of pregnant rats, and rabbits. On a mg/kg basis, the doses used were up to 333 times (in rats), and 30 times (in rabbits) the maximum recommended human dose.

**Hydrochlorothiazide, Teratogenic Effects:** Reproduction studies in the rabbit, mouse, and the rat at doses up to 100 mg/kg/day (50 times the human dose) showed no evidence of external abnormalities of the fetus due to hydrochlorothiazide. Hydrochlorothiazide given in a two-liter study in rats at doses of 4-5.6 mg/kg/day (approximately 1-2 times the usual daily human dose) did not impair fertility or produce birth abnormalities in the offspring. Thiazides cross the placental barrier and appear in cord blood.

**Nonteratogenic Effects:** These may include fetal or neonatal jaundice, thrombocytopenia, and possibly other adverse reactions which have occurred in the adult.

**PRECAUTIONS:** General, Enalapril Maleate: Impaired Renal Function: As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe congestive heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with angiotensin converting enzyme inhibitors, including enalapril, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20 percent of patients. These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy. In such patients renal function should be monitored during the first few weeks of therapy.

Some patients with hypertension or heart failure with no apparent pre-existing renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when enalapril has been given concomitantly with a diuretic. This is more likely to occur in patients with pre-existing renal impairment. Dose reduction of enalapril and/or discontinuation of the diuretic may be required.

**Evaluation of the hypertensive patient should always include assessment of renal function.**

**Hemodialysis Patients:** Anaphylactoid reactions have been reported in patients dialyzed with high-flux membranes (e.g., AN 69®) and treated concomitantly with an ACE inhibitor. In these patients consideration should be given to using a different type of dialysis membrane or a different class of antihypertensive agent.

**Hyperkalemia:** Elevated serum potassium (greater than 5.7 mEq/L) was observed in approximately one percent of hypertensive patients in clinical trials treated with enalapril alone. In most cases these were isolated values which resolved despite continued therapy, although hyperkalemia was a cause of discontinuation of therapy in 0.28 percent of hypertensive patients. Hyperkalemia was less frequent (approximately 0.1 percent) in patients treated with enalapril plus hydrochlorothiazide. Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with enalapril. (See Drug Interactions.)

**Cough:** Cough has been reported with the use of ACE inhibitors. Characteristically, the cough is nonproductive, persistent and resolves after discontinuation of therapy. ACE inhibitor-induced cough should be considered as part of the differential diagnosis of cough.

**Surgery/Anesthesia:** In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

**Hydrochlorothiazide:** Periodic determination of serum electrolytes to detect possible electrolyte imbalance should be performed at appropriate intervals. All patients receiving thiazide therapy should be observed for clinical signs of fluid or electrolyte imbalance: hyponatremia, hypochloremic alkalosis, and hypokalemia. Serum and urine electrolyte determinations are particularly important when the patient is vomiting excessively or receiving parenteral fluids. Warning signs or symptoms of fluid and electrolyte imbalance, irrespective of cause, include dryness of mouth, thirst, weakness, lethargy, drowsiness, restlessness, confusion, seizures, muscle pains or cramps, muscular fatigue, hypotension, oliguria, tachycardia, and gastrointestinal disturbances such as nausea and vomiting.

Hyperkalemia may develop, especially with brisk diuresis, when severe cirrhosis is present, or after prolonged therapy. Interference with adequate oral electrolyte intake will also contribute to hyperkalemia. Hyperkalemia may cause cardiac arrhythmia and may also sensitize or exaggerate the response of the heart to the toxic effects of digitalis (e.g., increased ventricular irritability). Because enalapril reduces the production of aldosterone, concomitant therapy with enalapril attenuates the diuretic-induced potassium loss (see Drug Interactions, Agents Increasing Serum Potassium).

Although any chloride deficit is generally mild and usually does not require specific treatment except under extraordinary circumstances (as in liver disease or renal disease), chloride replacement may be required in the

treatment of metabolic alkalosis.

Dilutional hyponatremia may occur in edematous patients in hot weather; appropriate therapy is water restriction, rather than administration of salt except in rare instances when the hyponatremia is life-threatening. In actual salt depletion, appropriate replacement is the therapy of choice.

Hyperuricemia may occur or frank gout may be precipitated in certain patients receiving thiazide therapy.

In diabetic patients dosage adjustments of insulin or oral hypoglycemic agents may be required. Hyperglycemia may occur with thiazide diuretics. Thus latent diabetes mellitus may become manifest during thiazide therapy.

The antihypertensive effects of the drug may be enhanced in the postsympathectomy patient.

If progressive renal impairment becomes evident consider withholding or discontinuing diuretic therapy.

Thiazides have been shown to increase the urinary excretion of magnesium; this may result in hypomagnesemia.

Thiazides may decrease urinary calcium excretion. Thiazides may cause intermittent and slight elevation of serum calcium in the absence of known disorders of calcium metabolism. Marked hypercalcemia may be evidence of hidden hyperparathyroidism. Thiazides should be discontinued before carrying out tests for parathyroid function.

Increases in cholesterol and triglyceride levels may be associated with thiazide diuretic therapy.

**Information for Patients, Angioedema:** Angioedema, including laryngeal edema, may occur especially following the first dose of enalapril. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician.

**Hypotension:** Patients should be cautioned to report lightheadedness especially during the first few days of therapy. If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician.

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure; patients should be advised to consult with the physician.

**Hyperkalemia:** Patients should be told not to use salt substitutes containing potassium without consulting their physician.

**Neutropenia:** Patients should be told to report promptly any indication of infection (e.g., sore throat, fever) which may be a sign of neutropenia.

**Pregnancy:** Female patients of childbearing age should be told about the consequences of second- and third-trimester exposure to ACE inhibitors, and they should also be told that these consequences do not appear to have resulted from intrauterine ACE-inhibitor exposure that has been limited to the first trimester. These patients should be asked to report pregnancies to their physicians as soon as possible.

**NOTE:** As with many other drugs, certain advice to patients being treated with VASERETIC is warranted. This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects.

**Drug Interactions, Enalapril Maleate, Hypotension—Patients on Diuretic Therapy:** Patients on diuretics and especially those in whom diuretic therapy was recently instituted, may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril. The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS.)

**Agents Causing Renin Release:** The antihypertensive effect of enalapril is augmented by antihypertensive agents that cause renin release (e.g., diuretics).

**Other Cardiovascular Agents:** Enalapril has been used concomitantly with beta adrenergic-blocking agents, methylglucosides, nitrates, calcium-blocking agents, hydralazine and prazosin without evidence of clinically significant adverse interactions.

**Agents Increasing Serum Potassium:** Enalapril attenuates diuretic-induced potassium loss. Potassium-sparing diuretics (e.g., spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia they should be used with caution and with frequent monitoring of serum potassium.

**Lithium:** Lithium toxicity has been reported in patients receiving lithium concomitantly with drugs which cause elimination of sodium, including ACE inhibitors. A few cases of lithium toxicity have been reported in patients receiving concomitant enalapril and lithium and were reversible upon discontinuation of both drugs. It is recommended that serum lithium levels be monitored frequently if enalapril is administered concomitantly with lithium. Hydrochlorothiazide, when administered concurrently the following drugs may interact with thiazide diuretics:

**Alcohol, barbiturates, or narcotics:**—potentiation of orthostatic hypotension may occur.

**Antidiabetic drugs (oral agents and insulin):**—dosage adjustment of the antidiabetic drug may be required.

**Other antihypertensive drugs:**—additive effect or potentiation.

**Cholestyramine and colestipol resins:**—Cholestyramine and colestipol resins bind the hydrochlorothiazide and reduce its absorption from the gastrointestinal tract by up to 85 and 43 percent, respectively. Thiazides may be administered two to four hours before the resin when the two drugs are used concomitantly.

**Corticosteroids, ACTH:**—intensified electrolyte depletion, particularly hypokalemia.

**Pressor amines (e.g., norepinephrine):**—possible decreased response to pressor amines but not sufficient to preclude their use.

**Skeletal muscle relaxants, nondepolarizing (e.g., tubocurarine):**—possible increased responsiveness to the muscle relaxant.

**Lithium:**—should not generally be given with diuretics. Diuretic agents reduce the renal clearance of lithium and add a high risk of lithium toxicity. Refer to the package insert for lithium preparations before use of such preparations with VASERETIC.

**Non-steroidal Anti-inflammatory Drugs:**—In some patients, the administration of a non-steroidal anti-inflammatory agent can reduce the diuretic, natriuretic, and antihypertensive effects of loop, potassium-sparing and thiazide diuretics. Therefore, when VASERETIC and non-steroidal anti-inflammatory agents are used concomitantly, the patient should be observed closely to determine if the desired effect of the diuretic is obtained.

**Carcinogenesis, Mutagenesis, Impairment of Fertility:** Enalapril in combination with hydrochlorothiazide was not mutagenic in the Ames microbial mutagen test with or without metabolic activation. Enalapril-hydrochlorothiazide did not produce DNA single strand breaks in an *in vitro* alkaline elution assay in rat hepatocytes or chromosomal aberrations in an *in vivo* mouse

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#### bone marrow assay

**Enalapril Maleate:** There was no evidence of a tumorigenic effect when enalapril was administered for 106 weeks to rats at doses up to 90 mg/kg/day (150 times the maximum daily human dose). Enalapril has also been administered for 94 weeks to male and female mice at doses up to 90 and 180 mg/kg/day, respectively, (150 and 300 times the maximum daily dose for humans) and showed no evidence of carcinogenicity.

Neither enalapril maleate nor the active diacid was mutagenic in the Ames microbial mutagen test with or without metabolic activation. Enalapril was also negative in the following genotoxicity studies: *in vitro* sister chromatid exchange with cultured mammalian cells, and the micronucleus test with mice, as well as in an *in vivo* cytogenetic study using mouse bone marrow.

There were no adverse effects on reproductive performance in male and female rats treated with 10 to 90 mg/kg/day of enalapril.

**Hydrochlorothiazide:** Two-year feeding studies in mice and rats conducted under the auspices of the National Toxicology Program (NTP) uncovered no evidence of a carcinogenic potential of hydrochlorothiazide in female mice (at doses of up to approximately 600 mg/kg/day) or in male and female rats (at doses of up to approximately 100 mg/kg/day). The NTP, however, found equivocal evidence for hepatocarcinogenicity in male mice.

Hydrochlorothiazide was not genotoxic *in vitro* in the Ames mutagenicity assay of *Salmonella typhimurium* strains TA 98, TA 100, TA 1535, TA 1537, and TA 1538 and in the Chinese Hamster Ovary (CHO) test for chromosomal aberrations, or *in vivo* in assays using mouse germinal cell chromosomes, Chinese hamster bone marrow chromosomes, and the *Aspergillus* sex-linked recessive lethal trait gene. Positive test results were obtained only in the *in vitro* CHO Sister Chromatid Exchange (clastogenicity) and in the Mouse Lymphoma Cell (mutagenicity) assays, using concentrations of hydrochlorothiazide from 43 to 1300 µg/mL and in the *Aspergillus nidulans* non-disjunction assay at an unspecified concentration.

Hydrochlorothiazide had no adverse effects on the fertility of mice and rats of either sex in studies wherein these species were exposed, via their diet, to doses of up to 100 and 4 mg/kg, respectively, prior to conception and throughout gestation.

**Pregnancy:** Pregnancy Categories C (first trimester) and D (second and third trimesters). See WARNINGS, Pregnancy, Enalapril Maleate, Fetal/Neonatal Morbidity and Mortality.

**Nursing Mothers:** Enalapril and enalaprilat are detected in human milk in trace amounts. Thiazides do appear in human milk. Because of the potential for serious reactions in nursing infants from either drug, a decision should be made whether to discontinue nursing or to discontinue VASERETIC, taking into account the importance of the drug to the mother.

**Pediatric Use:** Safety and effectiveness in children have not been established.

**ADVERSE REACTIONS:** VASERETIC has been evaluated for safety in more than 1500 patients, including over 300 patients treated for one year or more. In clinical trials with VASERETIC, no adverse experiences peculiar to this combination drug have been observed. Adverse experiences that have occurred, have been limited to those that have been previously reported with enalapril or hydrochlorothiazide.

The most frequent clinical adverse experiences in controlled trials were: dizziness (8.6 percent), headache (5.5 percent), fatigue (3.9 percent) and cough (3.5 percent). Adverse experiences occurring in greater than two percent of patients treated with VASERETIC in controlled clinical trials were: muscle cramps (2.7 percent), nausea (2.5 percent), asthenia (2.4 percent), orthostatic effects (2.3 percent), impotence (2.2 percent), and diarrhea (2.1 percent).

Clinical adverse experiences occurring in 0.5 to 2.0 percent of patients in controlled trials included: *Body As A Whole:* Syncope, chest pain, abdominal pain, *Cardiovascular:* Orthostatic hypotension, palpitation, tachycardia, *Digestive:* Vomiting, dyspepsia, constipation, flatulence, dry mouth, *Nervous System/Psychiatric:* Insomnia, nervousness, paresthesia, somnolence, vertigo, *Skin:* Pruritus, rash, *Other:* Dyspnea, gout, back pain, arthralgia, diaphoresis, decreased libido, hirsutism, urinary tract infection.

**Angioedema:** Angioedema has been reported in patients receiving VASERETIC (0.6 percent). Angioedema associated with laryngeal edema may be fatal. If angioedema of the face, extremities, lips, tongue, glottis and/or larynx occurs, treatment with VASERETIC should be discontinued and appropriate therapy instituted immediately. (See WARNINGS.)

**Hypotension:** In clinical trials, adverse effects relating to hypotension occurred as follows: hypotension (0.9 percent), orthostatic hypotension (1.5 percent), other orthostatic effects (2.3 percent). In addition syncope occurred in 1.3 percent of patients. (See WARNINGS.)

**Cough:** See PRECAUTIONS, Cough.

**Clinical Laboratory Test Findings, Serum Electrolytes:** See PRECAUTIONS.

**Creatinine, Blood Urea Nitrogen:** In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0.6 percent of patients with essential hypertension treated with VASERETIC. More marked increases have been reported in other enalapril experience. Increases are more likely to occur in patients with renal artery stenosis. (See PRECAUTIONS.)

**Serum Uric Acid, Glucose, Magnesium, and Calcium:** See PRECAUTIONS.

**Hemoglobin and Hematocrit:** Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3 g percent and 1.0 vol percent, respectively) occur frequently in hypertensive patients treated with VASERETIC but are rarely of clinical importance unless another cause of anemia coexists. In clinical trials, less than 0.1 percent of patients discontinued therapy due to anemia.

**Liver Function Tests:** Rarely, elevations of liver enzymes and/or serum bilirubin have occurred. Other adverse reactions that have been reported with the individual components are listed below and, within each category, are in order of decreasing severity.

**Enalapril Maleate—Enalapril** has been evaluated for safety in more than 10,000 patients. In clinical trials, adverse reactions which occurred with enalapril were also seen with VASERETIC. However, since enalapril has been marketed, the following adverse reactions have been reported: *Body As A Whole:* Anaphylactoid reactions (see PRECAUTIONS, Hemodialysis Patients); *Cardiovascular:* Cardiac arrest, myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypotension in high risk patients (see WARNINGS, Hypotension); pulmonary embolism and infarction, pulmonary edema, rhythm disturbances including atrial tachycardia and bradycardia, atrial fibrillation; hypotension; angina pectoris; *Digestive:* Ileus, pancreatitis, hepatic failure, hepatitis (hepatocellular [proven on rechallenge] or cholestatic jaundice), melena, anorexia, glossitis, stomatitis, dry mouth, *Hematologic:* Rare cases of neutropenia, thrombocytopenia and bone marrow depression, a few cases of hemolysis in patients with G-6-PD deficiency have been reported in which a causal relationship to enalapril cannot be excluded, *Nervous System/Psychiatric:* Depression, confusion, ataxia, peripheral neuropathy (e.g., paresthesia, dysesthesia); *Urogenital:* Renal failure, oliguria, renal dysfunction (see PRECAUTIONS), flank pain, gynecostasia; *Respiratory:* Pulmonary infiltrates, bronchospasm, pneumonia, bronchitis, rhinorrhea, sore throat and hoarseness, asthma, upper respiratory infection; *Skin:* Exfoliative dermatitis, toxic epidermal necrolysis, Stevens-Johnson syndrome, herpes zoster, erythema multiforme, urticaria, alopecia, flushing, photosensitivity; *Special Senses:* Blurred vision, taste alteration, anosmia, conjunctivitis, dry eyes, tearing.

**Miscellaneous:** A symptom complex has been reported which may include a positive ANA, an elevated erythrocyte sedimentation rate, arthralgia/arthritis, myalgia, fever, serositis, vasculitis, leukocytosis, eosinophilia, photosensitivity, rash and other dermatologic manifestations.

**Fetal/Neonatal Morbidity and Mortality:** See WARNINGS, Pregnancy, Enalapril Maleate, Fetal/Neonatal Morbidity and Mortality.

**Hydrochlorothiazide—Body as a Whole:** Weakness, *Digestive:* Pancreatitis, jaundice (intrahepatic cholestatic jaundice), sialadenitis, cramping, gastric irritation, anorexia, *Hematologic:* Aplastic anemia, agranulocytosis, leukopenia, hemolytic anemia, thrombocytopenia, *Hypersensitivity:* Purpura, photosensitivity, urticaria, necrotizing angitis (vasculitis and cutaneous vasculitis), fever, respiratory distress including pneumonitis and pulmonary edema, anaphylactic reactions, *Musculoskeletal:* Muscle spasm, *Nervous System/Psychiatric:* Restlessness, *Renal:* Renal failure, renal dysfunction, interstitial nephritis (see WARNINGS); *Skin:* Erythema multiforme including Stevens-Johnson syndrome, exfoliative dermatitis including toxic epidermal necrolysis, alopecia; *Special Senses:* Transient blurred vision, xanthopsia.

\* Based on patient weight of 50 kg.

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**Action:** Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalamic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

**Indications:** Yocon® is indicated as a sympatholytic and mydriatic. It may have activity as an aphrodisiac.

**Contraindications:** Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

**Warning:** Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

**Adverse Reactions:** Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.<sup>1,2</sup> Also dizziness, headache, skin flushing reported when used orally.<sup>1,3</sup>

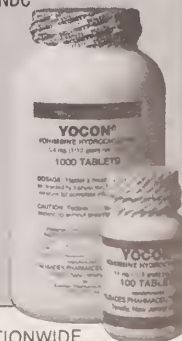
**Dosage and Administration:** Experimental dosage reported in treatment of erectile impotence.<sup>1,3,4</sup> 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to 1/2 tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.<sup>3</sup>

**How Supplied:** Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

#### References:

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# Sexually Speaking . . .

## Seasonal Affective Disorder: The hidden benefits

Mary B. Cavalier, M.S. <sup>(1)</sup>

The wonderful summer is becoming a distant memory as the trees change color and the snow slowly descends from the mountains. Patients are beginning to live in dread of the winter.

I have been noticing an interesting phenomenon associated with Seasonal Affective Disorder (SAD). I have been probing beyond the benign resignation to the impending doom. What I've been discovering is a "secondary gain" or benefit to seasonal affective disorder.

### SECONDARY GAIN TO SAD

The most stark example of a secondary gain or benefit became apparent while working with an incest survivor. The patient experiences severe depression in the winter. This winter he created a game plan which involved a lot of exercise. Procrastination has already set in. The depression is beginning to take hold.

Upon examining the procrastination, it was discovered that the last time the patient was physically active, he had a body memory of the incest. This, in turn, created an aversion to exercise. Therefore, when the patient's plan included large amounts of exercise, fear of the pains of the past triggered procrastination. SAD, or the avoidance of treatment for, enabled the patient to remain detached from his body and therefore from any further memories.

For another patient, SAD provided an opportunity to say "no." No to friend's invitations. No to family demands. No to her partner for sex. The depression enabled her to set limits where otherwise she doesn't feel safe. She felt understood when she blamed her lack of desire on the cold and darkness. To take action on recovering from SAD would mean she would also have to learn to be assertive.

### TREATMENT

The two cases presented above illustrate the range of secondary gain which can sabotage treatment for seasonal

affective disorder. As you well know, when a patient appears resistant to treatment, there usually is an underlying benefit to the discomfort.

I have found that a direct line of questioning has allowed the patient to arrive at the conclusion of self sabotage. I start off with an explanation of what is meant by secondary gain. This is then followed by questions such as, "What do you think the depression is protecting you from? What do you feel would happen if you were not depressed? What social responsibilities do you get to bow out of? What benefits do you think the depression is serving?"

A word of caution: This direct form of questioning doesn't work with patients who are clinically depressed. It does work for patients who, on the whole, function well and have a general sense of well-being. But, for whatever the secondary gain is, the patient avoids taking action on coping with the winter's darkness. When I've chosen to use such direct questioning with other patients, I have found that a well developed professional relationship is beneficial.

### CONCLUSION

With winter on its way, you may be finding yourself giving advice to those complaining of SAD. Suggestions such as exercise or use of full spectrum lights, etc. seem to be falling upon deaf ears. It may be helpful to confront the patient on the "benefits" he or she is experiencing as a result of the winter blues. The behavior may not change, but at least he or she can become more accountable for the lack of follow-through rather than complaining that "nothing" seems to help.

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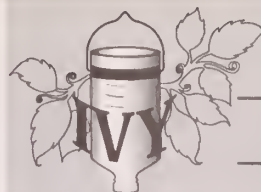
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# Methods of Reducing Obstetrical Claims

*Editor's Note: For purposes of this article, obstetricians include all physicians performing obstetrics.*

Neurological impairment of a neonate is a tragic outcome of pregnancy, and is particularly difficult to accept when there were no known clinical problems during the pregnancy. When the cause of an unfortunate outcome of pregnancy is unknown, emotional trauma often leads plaintiffs to believe that the physician was negligent, even if the standard of care was impeccable.

Damages awarded for obstetrics cases remain extremely high. The average indemnity paid for neurological deficit and other birth related injuries in newborns is \$350,000. Consequently, insurance premiums for OB-GYN are among the highest in the medical community.

Following are two practical guidelines that can help obstetricians reduce the possibility of losing a lawsuit after a disappointing outcome of labor and delivery. *The guidelines are simple: (1) save an umbilical cord sample on each delivery and run pH and blood gases if indicated, and (2) send the placenta to pathology if the gross exam reveals abnormalities or the pregnancy and/or newborn are abnormal.*

## QUALITY OF CARE

When an infant is born with neurological impairment, the obstetricians's care will be scrutinized. The following two methods of analysis may help rule out the question of malpractice by the obstetrician.

## Umbilical Cord and Blood Gas

In his article, "Umbilical Cord pH and Blood Gas Analysis," Gary D.V. Hankins, M.D., discusses the utility of cord blood gas determinations in assessing quality of obstetrical care and timeliness of intervention. He notes that Apgar scores "are widely misused and misapplied," (3) particularly with regard to birth asphyxia. The Apgar score, in fact, is not intended as a measure of assessment, but should be used as an index to the need for intervention by a pediatrician. (4)

Cord blood analysis provides an accurate measure of fetal pH, PO<sub>2</sub>, PCO<sub>2</sub>, concentration of bicarbonate, and buffer base deficit or excess at the time of birth.

"At the worst, umbilical pH and blood gas analysis will substantiate the already obvious - that the neonate was in poor condition at birth. Most often, however, it will show that the baby was born in a good metabolic state and that intrapartum care was appropriate." (3)

For each delivery, a doubly clamped segment of umbilical cord should be obtained and set aside. Specimens can be draw and analyzed as necessary. If the nursing staffs notified that a specimen will be routinely drawn, they will ensure that enough clamps are ready and available in the delivery room. Once a segment of cord is doubly clamped, pH and gas values are stable at room temperature for up to 30 minutes.

Dr. Hankins notes that because umbilical venous blood specimens principally reflect the maternal/placental circulation, while arterial blood reflects fetal condition, arterial blood is the preferred specimen for analysis. A mixture of arterial and venous blood is of minimal value. "Proper interpretation of cord blood results requires precise knowledge of the vessel of origin. A specimen containing a mixture of arterial and venous blood is very difficult to assess. Final values depend on the relative volume of blood collected from each vessel." (3)

Table 1 indicates normal values for pH, PCO<sub>2</sub>, PO<sub>2</sub>, bicarbonate and base deficit.

It should be noted that there is a wide range of "normal," and neonates with poor values can have high Apgar scores and excellent outcomes. The base deficit is the best estimate of metabolic acidosis and therefore the best measure of fetal well-being. (4)

## Placental Examination

Plaintiffs frequently attribute poor outcomes to the physician's failure to recognize and correct fetal hypoxia during labor and delivery. Injury or maldevelopment responsible for these outcomes has often taken place earlier in the pregnancy or neonatal period. Placental

**Table 1**

### Normal Values: Umbilical Cord Blood

Cord Blood	pH	PCO <sub>2</sub> (mm Hg)	PO <sub>2</sub> (mm Hg)	Bicarbonate (meq/L)	Base Deficit (meq/L)
Arterial	7.28±0.05	49.2±8.4	18.0±6.2	22.3±2.5	6.4±1.9
Venous	7.35±0.05	38.2±5.6	29.2±5.9	20.4±2.1	- -

Values are mean plus or minus standard deviations.(1)

exam may reveal the cause of preterm labor, premature membrane rupture, fetal undergrowth and antenatal hypoxia.(5)

When the pediatrician is called on to manage the care of an impaired neonate, he or she should use care in judging the cause of the damage. Too often neurological impairment is labeled as "birth asphyxia" when, in fact, the problem may have occurred before labor and delivery.

Here again, the obstetrician, pediatrician, and pathologist can join forces to discover possible causes of problems, and in many cases, to rule out medical malpractice. Pathologists with the ability to recognize pregnancy and neonatal disorders in the placenta may help to answer questions that might otherwise lead to malpractice claims.

In determining whether a pathologist needs to become involved the obstetrician should examine the placenta grossly. If one or more of the following indicators is noted, the placenta should be saved for analysis, even if the neonate appears normal:

### 1. Maternal Conditions

- \* Significant deficiency in prenatal care
- \* All hypertensive states and other preeclampsia
- \* Diabetes mellitus (including gestational glucose intolerance)
- \* Pre or peripartum fever
- \* Premature rupture of membranes (>24 hours)
- \* RH immunization/ ABO incompatibilities
- \* Polyhydramnios or oligohydramnios
- \* Infections: Herpes, Chlamydia, CMV, Rubella

### 2. Fetal Conditions

- \* Question of intrapartum distress:
  - Ominous FHR tracing
  - Meconium staining
- \* Low Apgar scores (e.g. 5 min. score of 7 or less)
- \* Early neonatal distress
- \* Multiple birth (single or fused placenta)
- \* Stillbirth or apparent congenital abnormalities
- \* Prematurity (<36 weeks) or Post-maturity (>42 weeks) or suspected IUGR or birthweight <1,500g
- \* Suspected infection

### 3. Placental Conditions

- \* All gross abnormalities of placenta and/or umbilical cord

The importance of providing adequate clinical information to the pathologist along with the placenta cannot be over emphasized. According to David Slater, M.D., a pathologist at Fresno Community Hospital and Medical Center, "this is the eternal plea of the pathologist; nowhere is it more valid than in placental pathology." (Correspondence with James Affleck, M.D.)

A recommended list of information to include with the placenta follows:

#### In all cases:

- \* Explanation of reason for the exam
- \* Brief review of pregnancy and delivery
- \* Apparent maturity in weeks
- \* Race
- \* Clinical gestational age
- \* Fetal weight

#### In cases of fetal distress:

- \* Notation as to timing of delivery and perinatal events, duration, severity, etc.

#### In cases of premature rupture of membrane:

- \* Time relative to delivery, suspected complications (if any)

Also, pathologists are encouraged to increase their confidence and competence in placental pathology. Dr. Slater notes that the range of "within normal limits" is ill-defined in some areas of placental pathology; individual experience, as well as familiarity with the work of recognized experts, are required to ensure meaningful results.

## PRENATAL ASSESSMENT

Of additional importance is prenatal assessment of the fetus, particularly in the last trimester. A check sheet helps prevent any omission of necessary care and provides an easy method of documenting care given.

Changing the estimated date of confinement (EDC) late in the pregnancy does not make good clinical practice, nor medical-legal sense. Many NORCAL claims revolve around post-date pregnancies wherein the original EDC has been changed late in the pregnancy. Robert Eden, M.D. states, "Definitive dates must be assigned early in pregnancy. Once estimated, by clinical examination or ultrasound biometry, these dates should not be revised."(2)

Serial examinations during the course of pregnancy should include measurement of uterine growth. Deviation from anticipated uterine growth warrants evaluation by sonography. Ultrasonography with biophysical profiles, stress and nonstress tests should be performed as indicated and results adequately documented.

Amniocentesis should be considered for recommended indications, and informed consent or refusal should be taken and documented. Finally, screening tests during pregnancy, as well as before, should be performed as indicated and results documented. Informed refusal of such tests should also be documented.



## CONCLUSION

While there are no guarantees, either in medicine or in malpractice allegations, physicians are not powerless. When obstetricians and pathologists work together and use the scientific methods available to them to help rule out malpractice in extremely difficult circumstances, these doctors are taking risk management into their own hands. Defense attorneys agree that even if the outcome of the cord blood or placental analysis is inconclusive, they would still prefer to have that information available when preparing the defense of a malpractice case. Physicians may not be able to avoid all claims, but they can reduce the likelihood of judgments for malpractice because of a poor outcome.

*Many thanks to David Slater, M.D. and Gerald Weiss, M.D., for their assistance with this article. And special thanks to James Affleck, M.D. and Ted Loring, M.D. for their contributions.*

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## President's Page

This is an exciting as well as an unsettling time to serve as president of our state medical association. Many of the issues which have been on the Association's agenda for years have come "front and center" for real this year: Health Care Reform; Tort Reform; and not the least, the identity of the Association itself.

ASMA representatives have been instrumental in drafting CHIPRA, and serving on the Health Access and Cost Containment Coalition to shepherd through the legislature meaningful solutions to Alaska's health care problems. As this goes to press, the Clinton Health Care Plan has gone to Congress in draft form, and your Association is working to shape a state plan which will meet the requirements of any federal mandate, while providing an "Alaskan" solution to this complex problem.

This is the year for meaningful Tort Reform in Alaska, so say those involved with this issue over the years. The combination of a receptive state legislature, and an active Alaskans for Liability Reform make reform seem more than just a fantasy. Your help -- letters, calls and dollars -- will be needed particularly after the legislative session resumes in January. Stay tuned . . .

While member involvement is high in the committee work of ASMA (approximately 90 members serve on at least one committee), some of our local medical societies are failing due to lack of participation. This poses a significant problem for the health of our state association which relies on our local societies for delegates, and prepares local society members for leadership positions within ASMA. Our House of Delegates and Board of Trustees will be addressing the issues of mission and participation this year.

Look for updates on all these issues in the "Heartbeat" and in future columns. I know the demands on all of us professionally continue to rise, however any time, suggestions, or constructive criticisms you have to share will make your association stronger and better able to work for you.

Shawn Hadley, M.D.  
President

*(continued from page 215)*

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# History of Medicine in Alaska

## Frank Pauls, DrPH

He claims to have been born in a bakery, the first of many his parents would own. In fact, he probably was delivered in an apartment behind the bakery in San Jose, California.

His father had emigrated from Germany; his mother was a first generation American. Oakland, where the next bakery was purchased, was a strong German community. Frank spoke a mixture of English and German until the United States entered World War I. It forced his parents to teach him English without German expressions. The bakery customers were suspicious of this toddler's talk since all things German were greatly suspect at this time.

A bakery equipment company conceived of selling its machinery by "granting franchises" for retail bakeries to grocery stores and markets. This company sent Frank's father to Honolulu in 1918 to assist a local grocer in establishing the first retail bakery in the Territory of Hawaii. The Pauls spent four years in Hawaii, an interval remembered with joy by young Frank because he had international playmates and only had to wear shoes on Sunday.

His memories are gleeful: riding street cars to test the limits of the ticket transfer; selling newspapers to tourists and learning the techniques of picking up coins with his toes. More seriously, he attended Punahou School. Every other year Frank and his mother went stateside. It was an idyllic way to be young.

His most horrific memory was being on a bridge in downtown Honolulu during the aftermath of a tidal wave. While the tsunami hit the far side of the island, the Honolulu harbor was drained dry. As the water rushed back into the harbor, small fishing boats rode the fast rising tide of water and some were carried beneath the bridge. Normally, the boats would lower their masts to pass under the bridge. But the incoming tide was so fast and high that some of the boats did not have time and the masts were sheared off as the boat swept under the bridge. One helmsman was decapitated.

In 1923, Frank's father was assigned to Yokohama, Japan but there was such anti-American feeling there that they went instead to Palo Alto. Frank's father liked to buy bakeries, get them going and then sell them. The Pauls owned the bakery here long enough for Frank to go to grade school and junior high. Virginia Wright O'Malley

was a classmate in elementary school.

The next bakery was in San Rafael where he graduated from high school. He then went on to Marin Junior College for two years and then to the University of California at Berkeley.

In San Rafael he had been influenced strongly by Dr. Beck, the part-time city health officer, who had advised him to go into public health which deals with communities of people and has far ranging opportunities for meaningful service.

He considered medical school. At the university, he entered the Department of Hygiene which had a two-year program in public health. All graduates of this program had to have solid knowledge of epidemiology, statistics, maternal and child health, sanitation, administration and public health law. In the laboratory, the students took first-year medical school courses.

With the passage of the social security act in the mid-1930s, many public health departments were established at the city, county and state levels, and there was a tremendous

demand for trained public health personnel. Universities were encouraged to set up short term programs to provide the training. At the University of California, the small faculty of the Department of Hygiene was expanded and some help was provided by its full-time students. The trainees were dubbed "ninety-day wonders."

After graduation, Frank Pauls served a six-month internship at Santa Clara County Hospital laboratory. He wryly observes that he was encouraged to go into the laboratory as a bacteriologist by a woman, his faculty advisor, Dr. Margaret Beattie, an outstanding public health microbiologist. She wanted men to enter the field. She felt that men would be more readily listened to and would upgrade benefits and salaries. Frank encountered many top notch female supervisors from whom he learned a lot.

He first worked at the Golden State Milk Company in San Francisco in their quality control laboratory. After six months, the chance came to work for the Bureau of Epidemiology, California State Department of Health. He was involved in interpreting blood smears for malaria and psittacosis control studies, involving much laboratory animal work.

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**Dr. Pauls was often the  
right man in the right place  
at the right time.**

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In March 1938, the opportunity came to work in Alaska.

Prior to 1936, the Territorial Board of Health provided public health services through the Commissioner of Health, W. W. Council, M.D. To cover the vast reaches of Alaska, three deputies had been commissioned to be in charge of Anchorage, Fairbanks and Nome. The deputies were paid \$25 a month; the Commissioner, who also covered Juneau, made \$50. The territorial legislature authorized Dr. Council to accept federal funds for a territorial health department defining the various functions to be covered. However, it took many years before there was legislative action establishing the department.

The public health laboratory in Juneau to which Frank came as a second microbiologist had been started by Maggie Oygard, Dr. Council's nurse-technician, and Warren Eveland, who bought his way out of the army, to serve as its chief. A secretary and dishwasher completed the staff.

This was virgin territory. Surveys were uncovering major health problems. Physicians used the laboratory as it was the only one in the territory. It was an exciting place as one never knew what the mail would bring. Perhaps a burlap sack of frozen wolf and dog heads sent in for rabies examination or a highly scented box of suspect foods.

Initial serologic surveys on food handlers were 20 percent positive for syphilis. In the Juneau-Sitka area there was a high incidence of primary syphilis with four to five positive darkfields a week. Many active cases were found in miners. The "girls on the line" knew when it was pay day and boated in on floating brothels.

An Aleutian island schoolteacher wired in a description of a strange rash. A Coast Guard cutter was dispatched on the two-day trip to the village. The ship's medic could not diagnose the rash but drew blood on over 100 people of all age groups. Ninety-five percent were syphilis positive. The BIA sent the cutter again, this time with a physician supplied with arsenicals. It took four months to arrest the outbreak. This Aleutian island epidemic was tracked to two young girls who had gone to Unalaska which was a very active port particularly in the fall when the Bering Sea cod fish fleet and the Alaska packer boats came in for shore leave.

However, the overwhelming problem was tuberculosis. One of the problems was to do a tb x-ray survey. The remoteness seemed to make this insurmountable, however the answer was to hire young physicians, among them Dr. Jack Haldemann and Dr. Wesson, to take portable Pickard x-ray units to the Bush by plane and dog sled. Therapy was frustrating since the BIA had only one hospital in Juneau with twelve beds. Tuberculosis was treated at the time only by bed rest. There were many cases of extrapulmonary tuberculosis including tuberculosis meningitis.

In 1940, there was a boom. Ladd Air Force Base was built and the Alaska Railroad was rebuilt. Anchorage grew from 3,000 to 15,000. A branch laboratory was necessary in Anchorage because of the lengthy turnaround. This was located on the fourth floor of the old Oddfellows Hall, still in existence next to the 4th Avenue Theater. It was a typical Alaska makedo. The Times operated ground floor front; Alaska Communication on the alley. A banquet and meeting room was on the second floor; Dr. Sutherland and a lawyer on the third floor. In the laboratory, some of the cabinets and shelves were built from packing crates. During the first six months, staff slept there. Traveling health department personnel slept there also.

During the war, the branch office in Anchorage grew. There were epidemics of typhoid fever, diphtheria, rabies, brucellosis, foodborne disease and water pollution. The Army used the serological and bacteriological services until their facilities were completed. The staff worked with the civil defense program in establishing "walking blood banks." Everyone was typed so that in an emergency a call could be made for the needed blood and donors would come in.

Things happened rapidly now for Frank Pauls. He had met Adelheid Guenther in Juneau where she was a child welfare worker. They were married in Anchorage in April 1941. Frank was commissioned in the Sanitary Corps of the Army in December 1943, the day their daughter, Cathy, was born. He went to England with the 125th General Hospital; Adelheid, a military dependent, returned to North Dakota.

When the war ended in Europe, his unit was tapped for the invasion of Japan. He returned to Alabama for training. After VJ Day, he was assigned to Harmon General Hospital in Texas where his family could join him. Laboratory officers were considered a critical medical specialty and he stayed in the service for four years serving at Camp Bowie and Camp Hood, Texas. The Pauls' son, Peter, now deceased, was born here.

After release from the Army, he went to the Harvard School of Public Health, earning a Master of Science. Then back to Anchorage where son, Chris, was born.

There was increasing concern over Alaska's tuberculosis and sanitation problems. Dr. C. E. Albrecht's impassioned plea to Congress for federal funds and assistance resulted in a rapid expansion of territorial health services. Mobile marine, highway and train health units covered the territory. Laboratory services were expanded and regional laboratories established in Fairbanks and Ketchikan.

The U.S. Public Health Service in the early 1950s established the Arctic Health Research Center in Anchorage with an emphasis on arctic health problems. The building on the corner of 6th and K St. housed it along with the regional laboratory so that common

facilities could be shared and costly duplication avoided. There was an excellent reference library. Dr. Jaek Haldemann became its first director.

The laboratory was active in both the Arctic Health Research Center and the Territorial Public Health Department. New techniques in fluorescent microscopy in the early diagnosing of streptococcal disease were studied, and a vaccine against rabies in animals was distributed. Again, the staff was increased.

In 1957, Frank was granted a two-year leave of absence for a World Health Organization assignment to Iran. He was to advise the Minister of Health of Iran as the head of a three-man international team on the establishment of a national public health laboratory center. Since Iran's health problems and need for laboratory services were similar to Alaska's, he found he could translate his Alaska experiences to the problems of Iran. Remote people have similar needs.

In 1963, he left Anchorage on a special Public Health Service program to earn a DrPH (Doctorate of Public Health) from the University of North Carolina.

After receiving his doctorate, he accepted appointments as assistant director of the state laboratory of hygiene in Madison and as assistant professor of preventive medicine at the School of Medicine, University of Wisconsin. For six years Adelheid and the children

remained in Anchorage where she was a memorable teacher of Latin. Fortunately, he was able to work with the Anthropology Department on an international biological program with studies on the Eskimo which got him back to Alaska on occasion.

In 1969, Dr. Pauls returned to Juneau as Chief of the Section of Laboratories. Later, he served as acting director of the Division of Public Health. He retired in 1979.

He is a Fellow of the American Public Health Association and a charter member of the Alaska Public Health Association, Alaska Society of Medical Technologists, Alaska Division American Society of Microbiologists and American Society for Circumpolar Health. Dr. Pauls thinks that it is important that the people being studied have a voice. The implementation of this policy has widened the program of the Circumpolar Health Conference.

Dr. Frank Pauls was often the right man in the right place at the right time. He took full advantage of these opportunities both in his education and with his chosen career. His has been a full life. Alaska is fortunate that he has spent so much of it here.

Gwynneth Gminder Wilson  
Alaska Medical Alliance

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# From Out of the Past. . . Over Thirty Years Ago

by Gloria K. Park, M.D.



Vol. 5 No. 2, June 1963



Single Track Vehicle

## SCIENTIFIC ARTICLES

*"Tuberculosis of Bones and Joints"* by Carroll Larson, MD - presented at the ASMA annual meeting.  
*"Peptic Ulcer and the Superior Mesenteric Artery Syndrome"* by Theodore Shohl, MD.  
*"Seal Oil Lipid Pneumonia in Eskimo Children"* by Thomas Harrison, MD (USPHS Kotzebue).  
*"Single Track Vehicle Injuries"* by Donald Kettelkamp, MD and William Mills Jr. MD.  
*"Poisonous Plants in Alaska"* by Christine Heller, PhD, on the staff of The Arctic Health Research Center.



Dr. Shohl

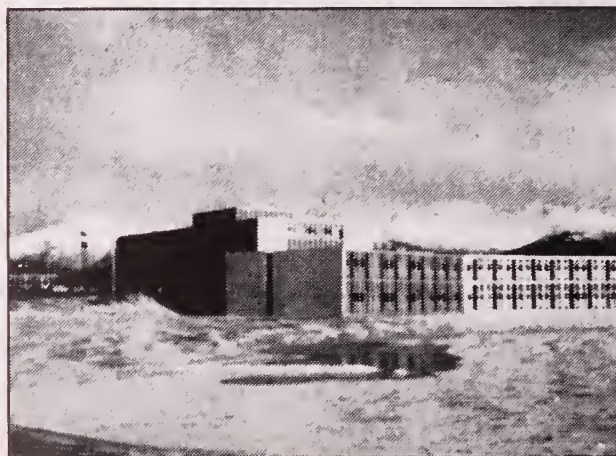
## EDITORIALS AND REPORTS

USPHS hospital has been approved for residency training in chest surgery. Three to four lung resections are done weekly, 90 percent for tuberculosis, 150 bronchoscopies per year due to high incidence of bronchiectasis.

API was opened October 16, 1962 and 50 patients were admitted in the first three months. Patients are also being transferred from Morningside in Portland, Oregon. There are over 100 employees.

ADH reports that clam digging for research purposes in June showed a toxin level the second highest in the past 30 years. It can cause peripheral and CNS aberrations that can reach total collapse.

Ketchikan General Hospital was dedicated August 11, 1963 with 80 beds. Following excerpts are from



Alaska Psychiatric Institute

Dr. Ralph Carr's presentation at the dedication. "The first hospital was about 1903 when Ketchikan was a pioneer community with dirt paths and wooden pathways. The present Hopkins Alley was Front Street. Mission Street was named because of St. John's Episcopal Mission. The St. John's Church Hospital was named the Arthur Yates Memorial Hospital. The Ketchikan General Hospital on Bowden Street opened in 1923. There was an episode in 1940 when Dr. Ronaldo Ellis and his associate Dr. Benny Rones tried to take an X-ray on a portable X-ray machine known to have a short circuit. Before Dr. Ellis had taken his hands off the machine, Dr. Rones pressed the button and the jolt of electricity knocked Dr. Ellis to the floor. Dr. Rones cried, 'I've killed him, I've killed him!' But Dr. Ellis came to, crawled in to a hospital bed for a day and a half and was soon all right. The Government during World War II built the improved obstetric delivery room on the second floor, added an operating room on the third; built the nurses' home, laundry and boiler room and added to each wing. Now in 1963 we have a fine new building near the Carlanna District. We should remember the early Ketchikan doctors such as Story, Carruthers, the three brothers Meyer, Beeson, Mustard, Ellis and Henry Turner."



Ketchikan General Hospital

## ASMA 18TH ANNUAL CONVENTION 1963

Members attending from:

ANCHORAGE — R. E. Harrell, Robert B. Wilkins, Wm. J. Mills, Jr., Joseph H. Shelton, Glenn Crawford, Robert Billings, James Fitzpatrick, James W. Coin, Ted Shohl, Wm. Rader, Rosalie Shohl, Don Val Langston, W. C. Matthews, George E. Hale, Alan Homy, Peter Koeniger, Virginia Wright, Frank Montmorency, Perry A. Mead, Gloria Park, James O'Malley, Charlie Chenoweth, Calvin Johnson, Winthrop Fish, Francis J. Phillips, John Tower, Chi Mei Chao, Elizabeth Tower, Louise Ormond, Thomas R. McGowan, Rudy

Leong, Helen Whaley, Robert Whaley, Royce H. Morgan, Milo H. Fritz, Thomas Keister, Mahlon Shoff, George Wichman, A. Claire Renn, Donald Kettelkamp, Duane L. Drake, Vernon A. Cates, Harold Bartko, Michael F. Beirne, Nancy Sydnam, Rodman Wilson, J. Ray Langdon.

BETHEL — Harriet J. Schirmer.

CHUGIAK — Marshall Simpson.

CORDOVA — W. John Chapman.

DILLINGHAM — John E. Libby.

FAIRBANKS — James Lundquist, Henry G. Storrs,

Joseph Ribar, Paul B. Haggland, George Clark, C.

William Bugh, Nicholas Deely, Donald E. Tatum.

GLENALLEN — Chester L. Schneider.

JUNEAU — Harry Akiyama, Grace E. Field, Levi M.

Browning, E. Stanley Ray, Edwin O. Wicks.

KETCHIKAN — Dwight L. Cramer, Ralph W. Carr.

KODIAK — J. Bruce Keers, R. Holmes Johnson

NASHVILLE, Tennessee — John A. Barrow, III.

NOME — Robert E. Fenstermacher.

SAND POINT — Carl E. Sandberg.

SEWARD — Joseph B. Deisher.

SITKA — Philip Moore, Edward D. Spencer.

SOLDOTNA — Paul G. Isaaks, Elmer E. Gaede.

SPENARD — William O. Rabourn.

Resolutions were passed regarding the Medical Practice Act, Mental Health planning commission, adoption, and X-ray standards. New officers elected were Donald Tatum, MD as President-elect; Robert Whaley, MD as Vice President; Robert Wilkins, MD as Secretary-Treasurer. Following excerpts are from the President's address (William J. Mills, Jr., MD) which included an historical review of ASMA. "In 1947, June 10, in the office of Dr. W.P. Blanton in Juneau, the Alaska Territorial Medical Association was incorporated. The Articles of Incorporation were written by Drs. Rude, Clements, Carter, Blanton, and Whitehead. Following incorporation the first Territorial Medical Association meeting was held in Fairbanks, in the chapel of the Mapleton Funeral Home. Dr. Paul Haggland was the first president pro tem and Dr. A. Holmes Johnson, president elect. Most of the business then was directed toward incorporation of the society and a scientific program was presented.

In 1948 a resolution was passed, introduced by Dr. Virginia Wright and Dr. James O'Malley, "opposing widespread commercialized prostitution in Alaska, and requesting legislation to formulate rules and regulations to prohibit the issuance of certificates to persons showing them to be free of venereal disease."

Dr. Earl Albrecht, Commissioner of Health, introduced a resolution for "legislation to provide the Department of Welfare with more financial support to see that the indigent sick in the state were well provided for."

Dr. Milo Fritz suggested a resolution "requesting



the medical meetings to be held at Juneau at the time of the next legislative session, in order to work with the legislature on matters of health."

In 1949 Dr. Albrecht presented a resolution directed to the federal government, recommending the establishment and maintenance of an Arctic Health Research Institute for this state, to be placed, if possible, in the region of the University of Alaska.

Further resolutions were adopted regarding the development of a Bureau of Vital Statistics, a pure food and drug act, and a recommendation that the health laws of the state be rewritten, with further clarification of the basic science regulations. At that time, at that meeting, a discussion was held regarding diploma mills in certain parts of the state of Alaska. Further discussion was had recommending an increase in salary for the office of Commissioner of Health. It is interesting that in 1949, in opposition to Mr. Ewing, it was proposed by Dr. Whitehead in Juneau, that a resolution be adopted opposing the program submitted by the President of the United States for national compulsory health insurance, but reiterating the stand of the previous year as being in favor of prepaid hospital care. Further interest in health care was demonstrated that same year with a resolution requesting the dentists to provide post-operative care for the treatment of patients following tooth extraction.

In 1950 the basic science laws were again discussed and need reiterated for their revision. The society went on record as recommending to the state and the Department of Civil Defense, the stockpiling of goods, including medical goods.

At that time, too, for the first time on record, or at least on record in our minutes, Dr. A. Holmes Johnson of Kodiak, Alaska, permitted a break in an otherwise impermeable wall, recommending that the wives of members of the Alaska Territorial Medical Association be encouraged to form a ladies auxilliary. This was passed unanimously.

In that same year, 1950, the society discussed drugless practitioners, advertising by paramedical individuals and radio and advertising claims for cure of cancer in the Southeastern area. It was recommended also that a public health committee be appointed by the Alaska Territorial Medical Association's president to consult and advise with the Department of Health of the Territory.

In 1951 a resolution was adopted encouraging the enlargement of the Pioneer Home facilities in Sitka, or elsewhere, to care for the aged. Dr. Whitehead of Juneau recommended investigation of the Florence Crittenden Homes and suggested that such a facility might be provided in the State of Alaska.

1952 a need was recognized for more hospitals in the Territory, and a resolution was passed to this effect. Dr. Walkowski was elected Physician of the Year, and further widened the breach in the dike by himself requesting that

the Women's Auxillary be continued as a permanent function of the Alaska Territorial Medical Association and suggested that they take part in all its social activities. This resolution was unanimously approved and adopted. Neither Dr. Johnson nor Dr. Walkowski has held office in the State Association since.

In 1953, in Sitka, interest in legislative matters was demonstrated when Dr. Blanton of Juneau suggested that the meeting in 1955 be held in Juneau to meet at the same time as the State Legislature. It was suggested further by Dr. Phillip Moore, that the Alaska Territorial State Medical Association be represented at the legislature during its session. A discussion was had of the "osteopathic, chiropractic, and other attendance law" passed by the Territorial Legislature as a rider. A further resolution was adopted recommending that the Alaska Mental Health Act, House Resolution 1217 be enacted, as the old act, the Organic Act, was archaic and inhumane and improper for Alaskans.

In 1954 further interest in medical legislation, particularly in regard to tuberculosis was demonstrated, and it was requested that more tuberculosis beds be provided for in the Territory. A discussion followed regarding "healers, reflexologists", and others using freely the prefix Dr. in advertising and on letterheads. It was noted that these individuals required only a business license to practice "medicine" and again it was decided that it was necessary to have representation on the floor of the Territorial House in Juneau to provide proper medical legislation for the Territory.

In 1955 there was considerable interest in regard to medical legislation by the Territorial Medical Association, including a recommendation for the establishment of health districts, the increase in health standards for trailer camps, interest in the presumptive death law, and further concern regarding mental health and its problems. Recommendations were made in favor of making prostitution itself a crime in Alaska; regulating the practice of physiotherapists in Alaska; and listing the educational requirements for chiropractors. There was interest by the society in House Bill 64, the Dental Practice Act, in which physicians were denied the right to practice dentistry in emergencies.

A very interesting discussion was had regarding the United States Public Health Service, and the transfer or potential transfer of PHS facilities to the State of Alaska. This action was triggered by a letter from the United States Public Health Service, Director of Indian Affairs, with a final letter by Gov. Heintzleman, indicating that it would not be feasible for the State of Alaska at that time and probably in the very far future, unless federal financial support was provided, and soon forthcoming.

In 1956 the Medical Association recommended legislation for the inspection of brakes, lights, and safety equipment on automobiles, private and commercial, for the betterment of the general public health. A resolution was adopted to appoint a physician on the Board of Public Welfare.

In 1957 a review of the Medical Practice Act was once more recommended, since changes in medical education had occurred, graduate requirements had increased, and it was known that all established forms and customs require review. This review was suggested for the more efficient operation of licensing procedures, and improvement of the medical care in the Territory.

In 1958 Dr. Phillips stimulated a discussion of the need for changes in the Medical Practice Act, particularly in regard to expediting the granting of licenses and temporary permits.

In 1959 the society met in Juneau at the Masonic Temple. A committee was appointed including Drs. Sparling, Moore, Whaley and Mills for study of the Maternal Child Health, Crippled Children's Service in the state. The committee was advised to confer with members of the legislature and the Alaska Department of Health in regard to these matters.

This conference was held, the members appeared before a legislative committee. Recommended legislation was not accepted, since at that time there was conflict between the members of the Alaska State Medical Association, particularly interested in that field, and the members of the Alaska Department of Health, over the need for such new legislation. This led, as you can imagine, to legitimate confusion in the ranks of the legislative body.

It was further recommended that there be established a division of Public Health within the Department of Health and Welfare, and the head of this division be a Director of Public Health, his position to be established by Governor's appointment.

That same year a resolution was passed recognizing the need for official representation of the Alaska State Medical Association with the legislature, this position to be established for the purpose of officially representing the State Society in Juneau at the time the legislature met. Dr. William Whitehead was appointed to this position. A further resolution was passed recommending the Governor appoint a physician member to the Atomic Energy Advisory Board, a member trained in radiologic health and safety procedures.

In 1960, three interesting resolutions in a row were passed regarding the Veterans Administration. The first, that the Veterans Administration limit its care program to veterans with service connected disability only. The second, that in view of the Veterans Administration's expenditure of almost one billion dollars a year, with 80 to 85 percent of the Veterans Administration's care for non service incurred disability, that the Veterans Administra-

tion dispense with its building program immediately. The third, that since government hospital facilities are already available that could be used for the care of veterans with service incurred disabilities, that the Veterans Administration hospitals be turned over to the states in which they are located to be used as the states see fit.

Again that year a committee was appointed to investigate the need for revision and modernization of the present Medical Practice Act, and if such need was found to exist, to work with the Board of Medical Examiners in revising the Act and requesting the legislature to make it law. Once more a resolution was adopted requesting the transfer of the Alaska Native Hospital System to the Alaska Department of Health and Welfare. Further recommendations were made regarding the development of foster homes and the care of children in those homes, since it was felt that children requiring long term hospitalization did better in foster homes than in hospitals.

In 1961 at Sitka, Dr. McBrayer in his opening address, recommended that legislation be enacted to increase the salaries of the professional personnel throughout the Department of Health and Welfare in order to obtain and keep men and women of high caliber in that department.

The society that year urged the Alaska State Legislature to pass the Kerr-Mills Bill and oppose the King-Anderson legislation. We went on record as opposing further, the bill by Senators Cooper, Gruening and Bartlett recommending restriction on the use of laboratory animals for medical research, and in effect opposed the antivivisection bill.

In 1962 Mr. Henry Camerot met with the Medical Association, having been the representative and advisor during the course of the legislature that year. A discussion was had of the adoption bill not passed, the professional incorporation bill, vetoed by the Governor, a marriage code, and a discussion of the bill on restriction of the use of x-ray by chiropractors, this bill returned to committee. The Kerr-Mills legislation and implementation bill was discussed and it was noted that this was not brought out of committee.

It was further recommended that a legislative representative be present physically in the capital city during the legislative sessions. This was done with good effect the past year. Our representative in Juneau is now Mr. Frank Doogan of the law firm of Faulkner, Banfield, Boechever and Doogan. The society reaffirmed the appointment of an advisory council to the Alaska Department of Health.

## **MUKTUK MORSELS:**

The oral polio immunization program reached over 60,000 in the Anchorage area, 10,000 in Fairbanks and an unknown number elsewhere.



The old Providence Hospital, now known as St. Mary's Residence has opened its doors as a facility for the chronically ill at a cost of \$20. per day to the patient.

The new Presbyterian Community Hospital in Anchorage is to open in June with 45 beds. Staff officers are Drs. George Hale, Vernon Cates, William Rader, A.S. Walkowski, Peter Koeniger and Howard Romig.

CORDOVA — Dr. Duane Drake of Anchorage was an eminently qualified judge for the Cordova Iceworm Festival Queen Contest.

KENAI — Dr. Paul Isaak accompanied by Dr. Charles St. John of Anchorage in the latter's Comanche traveled to a meeting in Denver. Return flight to Las Vegas and Sacramento was beautiful but the rest of the trip to Anchorage took a week with a series of smog and fog landings.

FAIRBANKS — Dr. Joe Ribar received the ASMA Community Service Award. Dr. Paul Haagland received the Silver Antelope award from the Boy Scouts. Three new physicians arrived in Fairbanks—Nicholas Deely, Joseph Johnson II, and Robert Faulkner.

HAINES — Dr. Philip Jones left and was replaced by Dr. Stanley Jones.

KETCHIKAN — Dr. Arthur Wilson and son Dr. James Wilson were joined by another son, Dr. Arthur Wilson Jr.

SANDPOINT — Dr. Carl Sandberg reports his new clinic is almost complete.

ST. PAUL — AHRC has been busy tracking simultaneous rubella and rubeola epidemics on the island.

JUNEAU — Two generalists have joined the Juneau clinic - Dr. John Dalton and Dr. G. Cummings Miller.

KANAKANAK — New MOC is Dr. Robert Fortuine.

KOTZEBUE — New MOC is Dr. Tom Harrison.

MT. EDGE CUMBE — New MOC is Dr. George Wagon.

PALMER — Dr. Walter Cunningham arrived and Dr. Arthur Colberg left for St. George Island.

ANCHORAGE — Dr. Robert Whaley received the Physician of the Year Award. A mountain climbing first was accomplished by Drs. George Wichman and Rod Wilson in reaching the summit of 12,600 ft. Mount Gerdine in the Alaska Range. Dr. Elizabeth Tower was one of 450 participants in the President Kennedy 50 mile hike from Palmer and was the 3rd woman to complete it. Dr. Nancy Sydnam has become a pilot. Dr. Gil Blankinship, formerly with the USPHS, has entered private practice. Also returning to Anchorage is Dr. Marcie Jackson and a new physician arriving is Dr. Warren Jones, formerly with the military in Fairbanks. Jack Hepler, MD and Keith Ryan, DDS were the objects of a CAP search when their float plane was damaged on a moose hunt.

### IN MEMORIAN:

*Cassins C. Carter, MD* — Died in Juneau due to brain cancer. Came to Tanana in 1932, to Juneau in 1934 and was a founder of the Juneau Clinic in 1936.

*Philip Cary Whitehead, MD* — Drowned in Seldovia Bay leaving his wife Betty Whitehead, MD and 5 children. They first came to Alaska in 1961.

*Dwight L. Cramer, MD* — Died suddenly in Ketchikan with a paralytic ileus. He settled in Ketchikan in 1937.

### CALL FOR PAPERS

The International Conference on Physician Health, to be held September 14-18, 1994 in Ottawa, Ontario, Canada is accepting abstracts addressing topics related to physician health, including AIDS, HIV, problems related to aging, mental illness, substance abuse, and physical disabilities and limitations, including those caused by general medical conditions. Possible topics for presentation include: presentation and treatment of health problems among physicians, the impact of disorders on physicians' families and practices, medical-legal issues facing hospital administrations and licensing boards, and material on health promotion and disease prevention. Abstracts which address issues related to these topics (i.e., prevention, diagnosis, treatment, rehabilitation), but not dealing specifically with physicians are also welcome. **Submission Deadline: February 1, 1994.**

Contact Elaine M. Tejcek, Department of Mental Health, American Medical Association, 515 N. State Street, Chicago, IL 60610, (312) 464-5073.

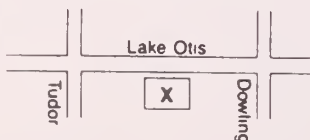
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# Glimpses of Alaskan Medical History

Edited by Robert Fortune, M.D.

## AN EXPLANATION OF THE HIGH MORTALITY AT NEW ARCHANGEL (1835-1840)

Blaschke's dissertation on Sitka is replete with morbidity and mortality statistics and his interpretation of them. A doctor of his time, he warmly subscribed to the idea that the pattern of disease in a locality was closely tied to the environment, especially the climate, precipitation, and winds. He discusses these ideas fully with respect to Sitka--an ideal spot, perhaps, to speculate on the changing weather and its effects.

In the following excerpt, Blaschke enumerates six reasons (unrelated to climate and geography, incidentally) why the mortality was unusually high at Sitka. Using Blaschke's figure for the 20-year average for the number of deaths at the capital, 31.1, divided by 800, a plausible estimate of the average number living at the capital over the previous two decades, we may calculate an annual mortality rate of 38.9/1000, over four times what one would expect today. These figures plainly show that life in Russian America was hard and tenuous, even where medical care was available.

The population of Sitka in 1841, according to Blaschke, was 981. Less than half (401) were Russians, a term he defines as also including Finns, Poles, Germans, and those from the Baltic States. Of the remainder, 20 were Siberian Natives known as Yakuts, 493 were Creoles, 51 were Aleuts (including persons from the Aleutians and from Kodiak Island), and 18 Koloshes, or Tlingit. Creoles were for the most part the offspring of Russian men and Aleut women, although the term has also been defined as a specific social class recognized by the Imperial Government. They were in many ways the mainstays of the work in the colonies, serving as feldshers, Orthodox priests, teachers, ship's captains, and in other important jobs. The Aleuts themselves were probably living in Sitka against their will, having been brought over from the islands as sea otter hunters. Finally, those Tlingit enumerated were only those dwelling in the Russian compound, with several hundred more living in the Indian village nearby.

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"Over a twenty-year period the total number of deaths was 622--a very remarkable number which exceeded the number of births by one-seventh. The source of this singular mortality, apart from endemic causes, is to be sought in the following: (1) In the health of those arriving and departing. Sick workers, some of them of advanced age and infirm, do not leave the colonies but remain there and are provided for at the expense of the Company. Those

enjoying such good health leave the settlement, but are often replaced by men who are not only advanced in age and feeble, but up to now more frequently sick, exhausted by the long journey and by the dissolute life they have lived during that time. Known everywhere by the name of Kamehadals, they are so despised in Siberia, at least, that often in entire districts they are unable to find a guest lodging. On the journey, however, they are provided with warm clothing and excellent food by the Company. (2) A more or less large number of sick persons, already for many years in the grip of chronic and often incurable diseases, come every year from the various colonial districts to Sitka as the capital. (3) Although ships which are to undertake longer voyages now have the benefit of surgeons, those intended for shorter journeys are provided with a kit of simple medications and instructions in what sort of cases and in what manner they are to be applied. Nevertheless, partly because of the difficulties inherent in shipboard life in treating the sick, and partly because of ignorance of treatment and the lack of assistance, those who have returned in these vessels are often deprived of all hope and will increase the mortality. (4) In the striking unexpected number of sudden deaths of those perishing from diverse accidents--men drowned while fishing and on other occasions or those killed in various types of work, especially that associated with ships, and also those dead of apoplexy from the abuse of alcohol. (5) In the extreme laxness of the women in matters of health and of their own life, and in their scorn for treatments and advice concerning their feeble constitution and consumptive nature. Finally (6) in the diseases of children proceeding from the laxness and inappropriate care given them by their mothers and with the shortage of wet nurses, on which account the rearing of infants is made very difficult and their preservation is rendered sometimes impossible, if for whatever reason if a mother is unable to lactate or if she is torn away by death. The average number of deaths per year is 31.1 (maximum 59, minimum 17)."

## REFERENCES

1. Blaschke, Eduard. *Dissertatio inauguralis sistens topographiam medicam Portus Novi-Archangelscensis, sedis principalis coloniarum rossicarum in Septentrionali America*. St. Petersburg: Wienhoeber and Sons, 1842. Pp. 41-42. (Translation by editor)



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## **PRAVACHOL® (Pravastatin Sodium Tablets)**

### **CONTRAINDICATIONS**

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Active liver disease or unexplained, persistent elevations in liver function tests (see WARNINGS).

**Pregnancy and lactation.** Atherosclerosis is a chronic process and discontinuation of lipid-lowering drugs during pregnancy should have little impact on the outcome of long-term therapy of primary hypercholesterolemia. Cholesterol and other products of cholesterol biosynthesis are essential components for fetal development (including synthesis of steroids and cell membranes). Since HMG-CoA reductase inhibitors decrease cholesterol synthesis and possibly the synthesis of other biologically active substances derived from cholesterol, they may cause fetal harm when administered to pregnant women. Therefore, HMG-CoA reductase inhibitors are contraindicated during pregnancy and in nursing mothers. **Pravastatin should be administered to women of child-bearing age only when such patients are highly unlikely to conceive and have been informed of the potential hazards.** If the patient becomes pregnant while taking this class of drug, therapy should be discontinued and the patient apprised of the potential hazard to the fetus.

### **WARNINGS**

**Liver Enzymes:** HMG-CoA reductase inhibitors, like some other lipid-lowering therapies, have been associated with biochemical abnormalities of liver function. Increases of serum transaminase (ALT, AST) values to more than 3 times the upper limit of normal occurring on 2 or more (not necessarily sequential) occasions have been reported in 1.3% of patients treated with pravastatin in the U.S. over an average period of 18 months. These abnormalities were not associated with cholestasis and did not appear to be related to treatment duration. In those patients in whom these abnormalities were believed to be related to pravastatin and who were discontinued from therapy, the transaminase levels usually fell slowly to pretreatment levels. These biochemical findings are usually asymptomatic although worldwide experience indicates that anorexia, weakness, and/or abdominal pain may also be present in rare patients.

As with other lipid-lowering agents, liver function tests should be performed during therapy with pravastatin. Serum aminotransferases, including ALT (SGPT), should be monitored before treatment begins, every six weeks for the first three months, every eight weeks during the remainder of the first year, and periodically thereafter (e.g., at about six-month intervals). Special attention should be given to patients who develop increased transaminase levels. Liver function tests should be repeated to confirm an elevation and subsequently monitored at more frequent intervals. If increases in ALT and AST equal or exceed three times the upper limit of normal and persist, then therapy should be discontinued. Persistence of significant aminotransferase elevations following discontinuation of therapy may warrant consideration of liver biopsy.

Active liver disease or unexplained transaminase elevations are contraindications to the use of pravastatin (see CONTRAINDICATIONS). Caution should be exercised when pravastatin is administered to patients with a history of liver disease or heavy alcohol ingestion (see CLINICAL PHARMACOLOGY: Pharmacokinetics/Metabolism). Such patients should be closely monitored, started at the lower end of the recommended dosing range, and titrated to the desired therapeutic effect.

**Skeletal Muscle:** Rhabdomyolysis with renal dysfunction secondary to myoglobinuria has been reported with pravastatin and other drugs in this class. Uncomplicated myalgia has also been reported in pravastatin-treated patients (see ADVERSE REACTIONS). Myopathy, defined as muscle aching or muscle weakness in conjunction with increases in creatine phosphokinase (CPK) values to greater than 10 times the upper limit of normal was reported to be possibly due to pravastatin in only one patient in clinical trials (<0.1%). Myopathy should be considered in any patient with diffuse myalgias, muscle tenderness or weakness, and/or marked elevation of CPK. Patients should be advised to report promptly unexplained muscle pain, tenderness or weakness, particularly if accompanied by malaise or fever. **Pravastatin therapy should be discontinued if markedly elevated CPK levels occur or myopathy is diagnosed or suspected. Pravastatin therapy should also be temporarily withheld in any patient experiencing an acute or serious condition predisposing to the development of renal failure secondary to rhabdomyolysis, e.g., sepsis; hypotension; major surgery; trauma; severe metabolic, endocrine, or electrolyte disorders; or uncontrolled epilepsy.**

The risk of myopathy during treatment with lovastatin is increased if therapy with either cyclosporine, gemfibrozil, erythromycin, or niacin is administered concurrently. There is no experience with the use of pravastatin together with cyclosporine. Myopathy has not been observed in clinical trials involving small numbers of patients who were treated with pravastatin together with niacin. One trial of limited size involving combined therapy with pravastatin and gemfibrozil showed a trend toward more frequent CPK elevations and patient withdrawals due to musculoskeletal symptoms in the group receiving combined treatment as compared with the groups receiving placebo, gemfibrozil, or pravastatin monotherapy. Myopathy was not reported in this trial (see PRECAUTIONS: Drug Interactions). One patient developed myopathy when clofibrate was added to a previously well tolerated regimen of pravastatin; the myopathy resolved when clofibrate therapy was stopped and pravastatin treatment continued. **The use of fibrates alone may occasionally be associated with myopathy. The combined use of pravastatin and fibrates should generally be avoided.**

### **PRECAUTIONS**

**General:** Pravastatin may elevate creatine phosphokinase and transaminase levels (see ADVERSE REACTIONS). This should be considered in the differential diagnosis of chest pain in a patient on therapy with pravastatin.

**Homozygous Familial Hypercholesterolemia.** Pravastatin has not been evaluated in patients with rare homozygous familial hypercholesterolemia. In this group of patients, it has been reported that HMG-CoA reductase inhibitors are less effective because the patients lack functional LDL receptors.

**Renal Insufficiency.** A single 20 mg oral dose of pravastatin was administered to 24 patients with varying degrees of renal impairment (as determined by creatinine clearance). No effect was observed on the pharmacokinetics of pravastatin or its 3 $\alpha$ -hydroxy isomeric metabolite (SQ 31,906). A small increase was seen in mean AUC values and half-life (t<sub>1/2</sub>) for the inactive enzymatic ring hydroxylation metabolite (SQ 31,945). Given this small sample size, the dosage administered, and the degree of individual variability, patients with renal impairment who are receiving pravastatin should be closely monitored.

**Information for Patients:** Patients should be advised to report promptly unexplained muscle pain, tenderness or weakness, particularly if accompanied by malaise or fever.

**Drug Interactions:** Immunosuppressive Drugs, Gemfibrozil, Niacin (Nicotinic Acid), Erythromycin: See WARNINGS: Skeletal Muscle.

**Antipyrine:** Clearance by the cytochrome P450 system was unaltered by concomitant administration of pravastatin. Since pravastatin does not appear to induce hepatic drug-metabolizing enzymes, it is not expected that any significant interaction of pravastatin with other drugs (e.g., phenytoin, quinidine) metabolized by the cytochrome P450 system will occur.

**Cholestyramine/Colestipol:** Concomitant administration resulted in an approximately 40 to 50% decrease in the mean AUC of pravastatin. However, when pravastatin was administered 1 hour before or 4 hours after cholestyramine or 1 hour before colestipol and a standard meal, there was no clinically significant decrease in bioavailability or therapeutic effect (see DOSAGE AND ADMINISTRATION: Concomitant Therapy).

**Warfarin:** In a study involving 10 healthy male subjects given pravastatin and warfarin concomitantly for 6 days, bioavailability parameters at steady state for pravastatin (parent compound) were not altered. Pravastatin did not alter the plasma protein-binding of warfarin. Concomitant dosing did increase the AUC and C<sub>max</sub> of warfarin but did not produce any changes in its anticoagulant action (i.e., no increase was seen in mean prothrombin time after 6 days of concomitant therapy). However, bleeding and extreme prolongation of prothrombin time has been reported with another drug in this class. Patients receiving warfarin-type anticoagulants should have their prothrombin times closely monitored when pravastatin is initiated or the dosage of pravastatin is changed.

**Cimetidine:** The AUC<sub>0-12h</sub> for pravastatin when given with cimetidine was not significantly different from the AUC for pravastatin when given alone. A significant difference was observed between the AUC's for pravastatin when given with cimetidine compared to when administered with antacid.

**Digoxin:** In a crossover trial involving 18 healthy male subjects given pravastatin and digoxin concurrently for 9 days, the bioavailability parameters of digoxin were not affected. The AUC of pravastatin tended to increase, but the overall bioavailability of pravastatin plus its metabolites SQ 31,906 and SQ 31,945 was not altered.

**Gemfibrozil:** In a crossover study in 20 healthy male volunteers given concomitant single doses of pravastatin and gemfibrozil, there was a significant decrease in urinary excretion and protein binding of pravastatin. In addition, there was a significant increase in AUC, C<sub>max</sub>, and T<sub>max</sub> for the pravastatin metabolite SQ 31,906. Combination therapy with pravastatin and gemfibrozil is generally not recommended.

In interaction studies with aspirin, antacids (1 hour prior to PRAVACHOL), cimetidine, niacin, or probucol, no statistically significant differences in bioavailability were seen when PRAVACHOL (pravastatin sodium) was administered.

**Other Drugs:** During clinical trials, no noticeable drug interactions were reported when PRAVACHOL was added to diuretics, antihypertensives, digitalis, converting-enzyme inhibitors, calcium channel blockers, beta-blockers, or nitroglycerin.

**Endocrine Function:** HMG-CoA reductase inhibitors interfere with cholesterol synthesis and lower circulating cholesterol levels and, as such, might theoretically blunt adrenal or gonadal steroid hormone production. Results of clinical trials with pravastatin in males and post-menopausal females were inconsistent with regard to possible effects of the drug on basal steroid hormone levels. In a study of 21 males, the mean testosterone response to human chorionic gonadotropin was significantly reduced (p<0.004) after 16 weeks of treatment with 40 mg of pravastatin. However, the percentage of patients showing a  $\geq$ 50% rise in plasma testosterone after human chorionic gonadotropin stimulation did not change significantly after therapy in these patients. The effects of HMG-CoA reductase inhibitors on spermatogenesis and fertility have not been studied in adequate numbers of patients. The effects, if any, of pravastatin on the pituitary-gonadal axis in pre-menopausal females are unknown. Patients treated with pravastatin who display clinical evidence of endocrine dysfunction should be evaluated appropriately. Caution should also be exercised if an HMG-CoA reductase inhibitor or other agent used to lower cholesterol levels is administered to patients also receiving other drugs (e.g., ketoconazole, spironolactone, cimetidine) that may diminish the levels or activity of steroid hormones.

**CNS Toxicity:** CNS vascular lesions, characterized by perivascular hemorrhage and edema and mononuclear cell infiltration of perivascular spaces, were seen in dogs treated with pravastatin at a dose of 25 mg/kg/day, a dose that produced a plasma drug level about 50 times higher than the mean drug level in humans taking 40 mg/day. Similar CNS vascular lesions have been observed with several other drugs in this class.

A chemically similar drug in this class produced optic nerve degeneration (Wallenian degeneration of retinogeniculate fibers) in clinically normal dogs in a dose-dependent fashion starting at 60 mg/kg/day, a dose that produced mean plasma drug levels about 30 times higher than the mean drug level in humans taking the highest recommended dose (as measured by total enzyme inhibitory activity). This same drug also produced vestibulocochlear Wallenian-like degeneration and retinal ganglion cell chromatolysis in dogs treated for 14 weeks at 180 mg/kg/day, a dose which resulted in a mean plasma drug level similar to that seen with the 60 mg/kg dose.

**Carcinogenesis, Mutagenesis, Impairment of Fertility:** In a 2-year study in rats fed pravastatin at doses of 10, 30, or 100 mg/kg body weight, there was an increased incidence of hepatocellular carcinomas in males at the highest dose (p<0.01). Although rats were given up to 125 times the human dose (HD) on a mg/kg body weight basis, their serum drug levels were only 6 to 10 times higher than those measured in humans given 40 mg pravastatin as measured by AUC.

The oral administration of 10, 30, or 100 mg/kg (producing plasma drug levels approximately 0.5 to 5.0 times human drug levels at 40 mg) of pravastatin to mice for 22 months resulted in a statistically significant increase in the incidence of malignant lymphomas in treated females when all treatment groups were pooled and compared to controls (p<0.05). The incidence was not dose-related and male mice were not affected.

A chemically similar drug in this class was administered to mice for 72 weeks at 25, 100, and 400 mg/kg body weight, which resulted in mean serum drug levels approximately 3, 15, and 33 times higher than the mean human serum drug concentration (as total inhibitory activity) after a 40 mg oral dose. Liver carcinomas were significant in high-dose females and mid- and high-dose males, with a maximum incidence of 90 percent in males. The incidence of adenomas of the liver was significantly increased in mid- and high-dose females. Drug treatment also significantly increased the incidence of lung adenomas in mid- and high-dose males and females. Adenomas of the eye Harderian gland (a gland of the eye of rodents) were significantly higher in high-dose mice than in controls.

No evidence of mutagenicity was observed *in vitro*, with or without rat liver metabolic activation, in the following studies: microbial mutagen tests, using mutant strains of *Salmonella typhimurium* or *Escherichia coli*; a forward mutation assay in L5178Y TK +/– mouse lymphoma cells; a chromosomal aberration test in hamster cells; and a gene conversion assay using *Saccharomyces cerevisiae*. In addition, there was no evidence of mutagenicity in either a dominant lethal test in mice or a micronucleus test in mice.

In a study in rats, with daily doses up to 500 mg/kg, pravastatin did not produce any adverse effects on fertility or general reproductive performance. However, in a study with another HMG-CoA reductase inhibitor, there was decreased fertility in male rats treated for 34 weeks at 25 mg/kg body weight, although this effect was not observed in a subsequent fertility study when this same dose was administered for 11 weeks (the entire cycle of spermatogenesis, including epididymal maturation). In rats treated with this same reductase inhibitor at 180 mg/kg/day, seminiferous tubule degeneration (necrosis and loss of spermatogenic epithelium) was observed. Although not seen with pravastatin, two similar drugs in this class caused drug-related testicular atrophy, decreased spermatogenesis, spermatocytic degeneration, and giant cell formation in dogs. The clinical significance of these findings is unclear.

**Pregnancy: Pregnancy Category X:** See CONTRAINDICATIONS.

Safety in pregnant women has not been established. Pravastatin was not teratogenic in rats at doses up to 1000 mg/kg daily or in rabbits at doses of up to 50 mg/kg daily. These doses resulted in 20x (rabbit) or 240x (rat) the human exposure based on surface area (mg/meter<sup>2</sup>). However, in studies with another HMG-CoA reductase inhibitor, skeletal malformations were observed in rats and mice. PRAVACHOL (pravastatin sodium) should be administered to women of child-bearing potential only when such patients are highly unlikely to conceive and have been informed of the potential hazards. If the woman becomes pregnant while taking PRAVACHOL (pravastatin sodium), it should be discontinued and the patient advised again as to the potential hazards to the fetus.

**Nursing Mothers:** A small amount of pravastatin is excreted in human breast milk. Because of the potential for serious adverse reactions in nursing infants, women taking PRAVACHOL should not nurse (see CONTRAINDICATIONS).

**Pediatric Use:** Safety and effectiveness in individuals less than 18 years old have not been established. Hence, treatment in patients less than 18 years old is not recommended at this time. (See also PRECAUTIONS: General.)

### **ADVERSE REACTIONS**

Pravastatin is generally well tolerated, adverse reactions have usually been mild and transient. In 4-month long placebo-controlled trials, 1.7% of pravastatin-treated patients and 1.2% of placebo-treated patients were discontinued from treatment because of adverse experiences attributed to study drug therapy; this difference was not statistically significant. In long-term studies, the most common reasons for discontinuation were asymptomatic serum transaminase increases and mild, non-specific gastrointestinal complaints. During clinical trials the overall incidence of adverse events in the elderly was not different from the incidence observed in younger patients.

**Adverse Clinical Events:** All adverse clinical events (regardless of attribution) reported in more than 2% of pravastatin-treated patients in the placebo-controlled trials are identified in the table below, also shown are the percentages of patients in whom these medical events were believed to be related or possibly related to the drug:

Body System/Event	All Events %		Events Attributed to Study Drug %	
	Pravastatin (N = 900)	Placebo (N = 411)	Pravastatin (N = 900)	Placebo (N = 411)
Cardiovascular				
Cardiac Chest Pain	4.0	3.4	0.1	0.0
Dermatologic				
Rash			1.3	0.9
Gastrointestinal				
Nausea/Vomiting	3.3	7.1	2.9	3.4
Diarrhea	6.2	5.6	2.0	1.9
Abdominal Pain	5.4	6.9	2.0	3.9
Constipation	4.0	7.1	2.4	5.1
Flatulence	4.0	7.1	2.7	3.4
Heartburn	4.0	7.1	2.0	0.7
General				
Fatigue	3.8	3.4	1.9	1.0
Chest Pain	3.7	1.9	0.3	0.2
Influenza	2.4*	0.7	0.0	0.0
Musculoskeletal				
Localized Pain	10.0	9.0	1.4	1.5
Myalgia	2.7	1.0	0.6	0.0
Nervous System				
Headache	6.2	3.9	1.7*	0.2
Dizziness	3.3	3.2	1.0	0.5
Renal/Genitourinary				
Urinary Abnormality	2.4	2.9	0.7	1.2
Respiratory				
Common Cold	7.0	6.3	0.0	0.0
Rhinitis	4.0	4.1	0.1	0.0
Cough	2.6	1.7	0.1	0.0

\*Statistically significantly different from placebo.

The following effects have been reported with drugs in this class

**Skeletal:** myopathy, rhabdomyolysis.

**Neurological:** dysfunction of certain cranial nerves (including alteration of taste, impairment of extra-ocular movement, facial paresis), tremor, vertigo, memory loss, paresthesia, peripheral neuropathy, peripheral nerve palsy.

**Hypersensitivity Reactions:** An apparent hypersensitivity syndrome has been reported rarely which has included one or more of the following features: anaphylaxis, angioedema, lupus erythematosus-like syndrome, polymyalgia rheumatica, vasculitis, purpura, thrombocytopenia, leukopenia, hemolytic anemia, positive ANA, ESR increase, arthritis, arthralgia, urticaria, asthenia, photosensitivity, fever, chills, flushing, malaise, dyspnea, toxic epidermal necrolysis, erythema multiforme, including Stevens-Johnson syndrome.

**Gastrointestinal:** pancreatitis, hepatitis, including chronic active hepatitis, cholestatic jaundice, fatty change in liver, and, rarely, cirrhosis, fulminant hepatic necrosis, and hepatoma; anorexia, vomiting.

**Reproductive:** gynecomastia, loss of libido, erectile dysfunction.

**Eye:** progression of cataracts (lens opacities), ophthalmoplegia.

**Laboratory Test Abnormalities:** increases in serum transaminase (ALT, AST) values and CPK have been observed (see WARNINGS).

Transient, asymptomatic eosinophilia has been reported. Eosinophil counts usually returned to normal despite continued therapy. Anemia, thrombocytopenia, and leukopenia have been reported with other HMG-CoA reductase inhibitors.

**Concomitant Therapy:** Pravastatin has been administered concurrently with cholestyramine, colestipol, niacin, acid, probucol and gemfibrozil. Preliminary data suggest that the addition of either probucol or gemfibrozil to therapy with lovastatin or pravastatin is not associated with greater reduction in LDL-cholesterol than that achieved with lovastatin or pravastatin alone. No adverse reactions unique to the combination or in addition to those previously reported for each drug alone have been reported. Myopathy and rhabdomyolysis (with or without acute renal failure) have been reported when another HMG-CoA reductase inhibitor was used in combination with immunosuppressive drugs, gemfibrozil, erythromycin, or lipid-lowering doses of niacinic acid. Concomitant therapy with HMG-CoA reductase inhibitors and these agents is generally not recommended. (See WARNINGS: Skeletal Muscle and PRECAUTIONS: Drug Interactions.)

### **OVERDOSAGE**

There have been no reports of overdoses with pravastatin.

Should an accidental overdose occur, treat symptomatically and institute supportive measures as required. (J4-422A)

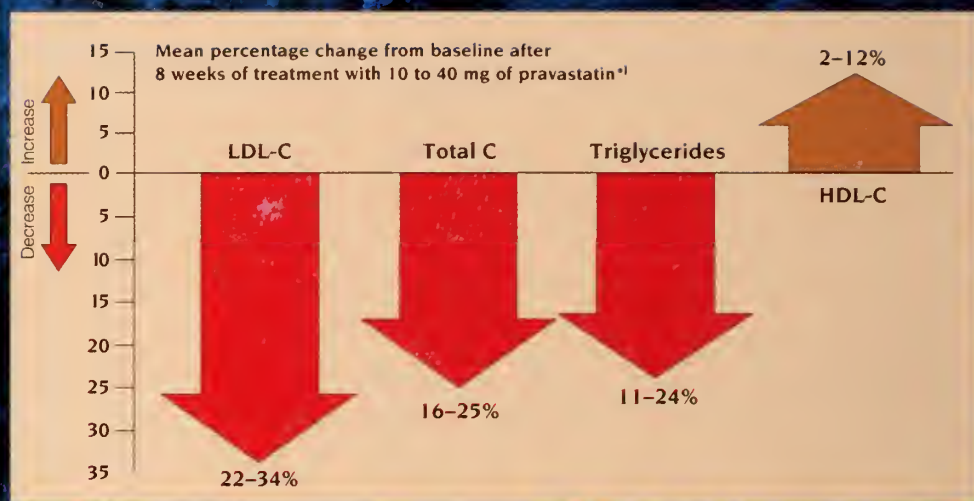




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**Reference:** 1. Jones PH, et al. Once-daily pravastatin in patients with primary hypercholesterolemia: a dose-response study. *Clin Cardiol*. 1991;14:146-151.

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Please see CONTRAINDICATIONS, WARNINGS, PRECAUTIONS, and ADVERSE REACTIONS in the brief summary of prescribing information on the adjacent page.



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About the cover: Child in Native Fur Parka. Photo courtesy of Lael Morgan/Alaska Magazine File Photo.



# CANCER IN INDIAN COUNTRY: A NATIONAL CONFERENCE

**Sponsored by:**  
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**Supported by:**  
**National Cancer Institute & The Network for Cancer Control Research among**  
**American Indian/Alaska Native Populations**

## ***ALASKA MEDICINE*** **Editor's Note —**

In this issue of *Alaska Medicine*, the proceedings of the National Conference "Cancer in Indian Country" which was held in Rapid City, South Dakota on September 15-17, 1992, will be presented. You will note that one of the papers is by Dr. Anne Lanier who is a recognized expert on the subject of cancer in Alaska Natives. Other groups of native Americans have health problems in common with Alaskans, other problems are unique.

We hope you find these papers intriguing.

Donald R. Rogers, M.D.  
Editor, *Alaska Medicine*

This issue of *Alaska Medicine* includes select presentations from a meeting "Cancer in Indian Country" held in September 1992. This meeting was significant in that it was only the second conference ever held in the U.S. on this subject. Cancer is now THE LEADING cause of death among Alaska Native women, and vies for second in Alaska Native men. Cancer death rates in Alaska Natives and Indians in the northern U.S. now *exceed* average U.S. rates.

These facts document a dramatic change since the 1950s when: cancer ranked seventh among causes of death, and the perception of medical providers was that this disease was rare in the Native populations. In Canada in 1952, the diagnosis of cancer in one Eskimo patient was thought to be so unusual it was reported in the journal "*Cancer*".

Cancer mortality data indicate that rates for all cancers and for various cancer sites differ widely among the various American Indian/Alaska Native (AI/AN) populations. This makes it difficult to generalize about the problem of cancer. However, the conference highlighted similarities as well as differences of importance to all involved with cancer prevention and control in AI/AN. These include: the widespread distribution of the population far from sophisticated cancer screening and treatment facilities; perceptions of causes of illness (especially cancer), and even taboos about cancer; the lack of a word for cancer in many Native languages; cultural and environmental barriers to screening and treatment; and the high prevalence of cancer risk factors in the populations.

The purpose of the meeting was to share knowledge and experience of those involved in control of this newly emerging problem in AI/AN. Proceedings of the first conference were published in the "*American Indian Culture and Research Journal*" (November 1992).

The Network for Native American Cancer Control Research among American Indians/Alaska Natives wishes to thank "*Alaska Medicine*" for publishing these proceedings and thus making the information available to the medical community.

Anne Lanier, M.D., M.P.H.  
Epidemiologist  
Alaska Area Native Health Service  
U.S. Public Health Service

# OVERVIEW: CANCER IN INDIAN COUNTRY — A NATIONAL CONFERENCE

Jennie R. Joe, Ph.D., M.P.H.  
Robert S. Young, Ph.D.

## A. THE PROBLEM OF CANCER IN AMERICAN/ALASKA NATIVES

Cancer is the third (second in women) leading cause of death in Native Americans; however, as Justice (1) has observed, information on cancer in Native Americans is limited in scope, and research has been conducted only on a few of the 510 recognized tribes. For example, in a comprehensive bibliography on cancer studies in Native Americans compiled for the years 1800-1989, Justice (2) found only 84 articles in which cancer is the primary subject.

Despite the fragmented and incomplete information on incidence, prevalence, and mortality from cancer for American Indians/Alaska Natives, a data base is emerging. This data base indicates that 1.) the proportion of site-specific neoplasms varies considerably from tribe to tribe (1); 2.) childhood cancer appears to be lower for American Indians/Alaska Natives (AI/AN); 3.) data on cancer for AI/AN remain troublesome because of small numbers, large confidence intervals, and inadequate recording of data; 4.) many types of cancers appear to be on the increase, and 5.) survival rates (33.4%) remain poor for most AI/AN diagnosed with cancer compared with the general population (51.4%) (3).

The cause for the lower cancer survival rates for AI/AN is not clear, but most observers link this poor outcome to late detection, inadequate access to health care, poverty, and various social and cultural barriers (4). Unfortunately, little information is available about some of these barriers, such as knowledge and attitudes of Native Americans about cancer. To date, much of what is known about cultural beliefs about cancer has been anecdotal. For example, Medicine (5) has said that her tribe, the Sioux, view cancer as a "white man's disease," and therefore cancer is viewed as a disease inflicted on the Indian by the Europeans. Jackson (6), a Navajo nurse, states that cancer to many of her tribal members is viewed as a fatal disease, and as a result cancer patients and/or their families may not seek treatment or other interventions. Wilson (7), a nurse practitioner and educator, has surmised that the fatalistic attitude toward cancer among the Navajos influences health care priorities, i.e., tribal health leaders fail to give priority to cancer prevention because they do not believe that "something can be done" about cancer.

Most of the American Indian and Alaska Native languages also have no word for cancer, and translations may lead to further misunderstandings about the disease. For example, among the Yup'ik (Eskimos) and the Navajos, cancer is referred to as "a sore that does not heal." By focusing on only one of the "symptoms" of cancer, it psychologically "sets the stage" for a fatalistic prognosis. Because not all neoplasms are visible to the eye and the pathological process of cancer is not always comprehended, cancer treatments may similarly be misunderstood. Misunderstanding is also fueled by the general fear of cancer.

Cancer is culturally one of the most dreaded diseases in mainstream society, and American Indians/Alaska Natives also share this fear. The fear, however, is often complicated by other socio-cultural beliefs about wellness and unwellness. For example, many tribes believed that "talking about an illness can bring about the illness." Thus there may be strong taboo about the "power" of words, and this perception may contribute to the fact that cancer may not be discussed in public and/or communities may not take preventive measures that would acknowledge anticipation of the disease. Thus various personal and cultural barriers delay early detection and/or may hamper various cancer prevention strategies.

The poor cancer survival rates obviously reflect some of this fatalistic attitude, but in most cases these deaths are also attributable to late diagnosis and, consequently, unsuccessful treatment outcomes. It is also important to note that because cancer is not publicly acknowledged in most reservation and urban Indian communities, there is a lack of support groups and/or opportunities for cancer survivors to provide support and encouragement to a newly diagnosed person with cancer. Moreover, early detection is a problem because preventive care is not available except in a few cases, e.g., routine cervical Pap smears for Indian women of child-bearing-age. Thus, there is a critical need to educate AI/AN communities as well as their health care providers about cancer, its cultural meanings, and its impact on the Indian communities.



## B. SIGNIFICANCE OF THE CONFERENCE

Because of the paucity of research data and information about cancer in Native Americans, the Native American Research and Training Center (NARTC) in 1989 sponsored the first research conference on cancer and Native Americans. The 1989 conference enabled researchers and clinicians working with Native Americans to meet and to discuss their cancer research activities — what has been done, what was currently in progress, and what future research directions should be emphasized. A “Proceedings” from this conference was published as a special supplement in the September 1992 issue of the *American Indian Culture and Research Journal* (8).

The National Cancer Institute (NCI) also convened and organized a Network for Cancer Control Research among American Indian and Alaska Native populations (Network) in 1989. In 1991, Network members decided to host a second cancer conference with a special focus on specific cancer control issues in the American Indian/Alaska Native populations for a cross section of researchers, tribal health leaders, and clinicians and other health care providers. The conference was also intended to promote a dialogue between traditional Indian medicine people and western medicine as a way to promote a collaborative agenda in the area of cancer prevention, control, and treatment.

## C. SPECIFIC AIMS OF THE CONFERENCE

The purpose of the conference was to address cancer research and prevention among American Indians/Alaska Natives. The specific goal of the conference was to provide information and updates for researchers, clinicians, service providers, traditional healers, and tribal health leaders on cancer prevention, control, and treatment. The objectives of the conference were as follows:

1. To convene a forum for researchers, clinicians, service providers, traditional healers, and community health leaders to present and exchange research information about cancer in AI/AN.
2. To provide updates on the scope of the problems of cancer, treatments, and prevention and control for new researchers, service providers, and tribal health leaders.
3. To compare with other minority populations, national and regional cancer data on AI/AN and to explore implications this data has for cancer prevention and control in this population.
4. To explore ways that western medicine and

traditional Indian medicine can work together in cancer prevention and control.

5. To identify and present culturally-relevant innovative intervention and prevention models in poster sessions, panels, and workshops.
6. To make abstracts and a current bibliography on cancer in AI/AN available to all registrants.
7. To produce a proceedings from selected papers and presentations that will serve as a reference for researchers and service providers.

The content of the conference included the following:

1. A review of pertinent epidemiological and other research data on cancer in AI/AN populations and a discussion of research needs and priorities.
2. Presentations and poster sessions that showcased model programs that help raise the level of cancer awareness among tribal health leaders, traditional healers, and health care providers.
3. Use of panel discussions and workshops that identified barriers and problems of access to health care for cancer patients in this population.
4. A review of existing information and research on the knowledge, attitudes, and beliefs about cancer as determined through research and as expressed by a panel comprising Native American cancer survivors.
5. An examination of issues of culture change (e.g., diet) and its relation to cancer prevalence and incidence in this population.

## D. CONFERENCE OUTCOMES

Nine major presentations at this conference were selected for inclusion as a “Proceedings.” These papers are grouped into the following four categories: (1) Epidemiology, (2) Risk Factors, (3) NCI research initiatives, and (4) Cultural considerations.

### Epidemiology

A number of presentations discussed the epidemiology of cancer in Native Americans and Alaska Natives by region. The two papers presented here as part of the “Proceedings” include Dr. James Hampton’s keynote address, which provided an overview of the problem of cancer in Native American peoples, and Dr. Anne Lanier’s discussion of the epidemiology of cancer in Alaska

Natives. As Hampton points out, although total age and sex adjusted rates for cancer are lower among American Indians (particularly rates among children) than in the general U.S. population, rates of some site specific cancers (stomach, liver, gall bladder, and cervix uteri) are much higher in American Indians. Furthermore, site specific cancer incidence varies by tribe and by region. Thus Plains Indians have high rates of lung cancer but low rates for melanoma; the reverse is true for the Tohono O'odham, a southwestern tribe which has high rates of melanoma and relatively low rates of lung cancer (1). Hampton also points out that in general cancer mortality rates are higher among American Indians/Alaska Natives than among the U.S. anglo population. Hampton also notes that poor five year survival rates have been observed for American Indians with the following cancers: stomach, colorectal, gall bladder, lung, breast, non-Hodgkin's lymphoma, liver, pancreas, and prostate. Total cancer incidence rates among Alaska Natives are similar to the U.S. all races rate when adjusted for sex and for age.

Lanier points out in her presentation that survival rates from cancer in this population are very low and attributes the poor survival rates to stage at diagnosis, refusal of treatment, and other environmental and host factors. Site specific cancers with extremely high incidence in Alaska Natives include: lung, nasopharynx, salivary gland, esophagus, stomach, liver, gall bladder, cervix, and kidney. In this presentation, Lanier also discusses those factors that make cancer prevention and control difficult in Alaska Native communities. Because one-third of all cancers in this population are tobacco related, Lanier recommends that cancer control efforts should focus on eradication of tobacco use.

## **Risk Factors**

Risk factors for cancer, including tobacco use, are the focus of papers by Dr. Thomas Welty and by Dr. Thomas Becker and their respective research groups. In a study of cancer among Plains Indian tribes, Welty and his group report that cancer mortality rates for Indians in the IHS Aberdeen area exceed U.S. rates, all races. The authors note that lung cancer is the leading cause of mortality due to cancer, and that cervical cancer is also 4.4 times higher than U.S. rates, all races. Welty and his group have also conducted a health risk appraisal and found high rates of tobacco use among both sexes. Rates for lung cancer are much lower in southwestern tribes because rates of tobacco are lower than the rates for the Plains tribes. The researchers recommend that cancer prevention programs be tailored to the cancer risk profile of the respective community in order to reduce preventable cancer deaths.

Dr. Thomas Becker and his research group have also found high rates of cervical cancer and dysplasia among

southwestern Native American women. Preliminary findings from a pilot study of risk factors show that cigarette smoking, HPV infection, use of contraceptives, parity, and a high number of sex partners are associated with dysplasia in Native American women. Significantly, these risk factors for cervical dysplasia appear to be different from those risk factors for cervical dysplasia identified for Hispanic and non-Hispanic white women.

## **NCI Cancer Research Initiatives For Native Americans**

Recognizing the seriousness of the problem of cancer in American Indian peoples, the NCI has since 1987 funded a number of initiatives for cancer prevention and intervention programs. Dr. Linda Burhansstipanov, a Project Officer with NCI, describes the history of NCI involvement in prevention and intervention programs for Native American groups. The author describes the specific NCI funded projects proposed by a number of groups that have developed and implemented prevention and intervention projects for breast and cervical cancer, and prevention projects that target tobacco and dietary risk factors for cancer. Burhansstipanov also describes a number of other program initiatives for Native American and Alaska Native groups and communities that have been funded by NCI.

The preliminary results of a number of the projects recently funded by NCI were presented at the conference. Two of these projects have been included in the "Proceedings." Leslie Cunningham-Sabo and Sally Davis describe a health promotion and cancer prevention project for fifth grade and seventh grade American Indian youth attending a number of schools in New Mexico. They note that rates of tobacco use by Native American adolescents, both smoking and chewing, are extremely high. In addition, there is a significant incidence of obesity in this population. Called "Pathways to Health," this cancer prevention program is designed to promote a healthy lifestyle using a culturally sensitive and meaningful program format.

The second project that was presented was the North Carolina Native American Cervical Cancer Prevention Project (NCP), a 5-year cervical cancer prevention screening program for rural Lumbee and Cherokee Indian women. According to Mark Dignan and his group, the cervical cancer mortality rate among these women is four times the rate among U.S. women, all races. Because of problems with the effectiveness of other education programs for Indian peoples, the NCP designed a model program that integrated Social Learning Theory and self-efficacy theory with a number of model education programs. The NCP is currently in its



third year of funding, and the investigators report the preliminary results of their program evaluation.

## Cultural Considerations

The element of culture is vital to the success of cancer prevention and intervention programs. Thus a number of presentations in the conference focused on cultural issues and beliefs about cancer and how these cultural issues impact on programs. Diane Weiner's presentation focused on cancer causation beliefs of the Luiseño Indians in southern California. In general, Weiner found that some cancer patients attributed their illness to heredity; others attributed their illness to malevolent sources or social transgressions (including sorcery or witchcraft) of other members of the community. The author also conducted interviews with individuals who had not had cancer. In contrast to the beliefs about cancer causation expressed by cancer patients,

these individuals attributed cancer to external forces such as pollution or to medical care "gone awry."

To add a human dimension to the statistics and the reports about programs, the Network planning committee invited a panel of American Indians who were cancer survivors to present their perspectives about the problems they experienced in their tribal communities. The panel comprised two cancer survivors and the widow of a cancer patient. As part of this "Proceedings," Judith Kaur, the moderator of the panel, has summarized some of the issues and problems described by these patients. Each panelist presented her respective medical history and then discussed the various social and cultural issues that the patient with cancer has to deal with. For example, one panelist noted that in her tribe that cancer was perceived as "contagious," thus pointing to the need for community wide education programs while at the same time indicating some of the barriers to implementing these kinds of programs in Indian communities. This panelist also discussed the resistance from the tribal council to her forming a support group of cancer patients and stressed the importance of such groups for the newly diagnosed cancer patient.

The authors of the papers included in this "Proceedings" all stress that the problem of cancer continues to increase in Native American communities. They call for health education programs about the disease and its risk factors and for improved access to cancer screening and treatment. Finally, they also stress the need to recognize cultural issues and barriers that must be overcome if health care providers are to be successful in implementing appropriate education and prevention programs that will have a significant impact.

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# CANCER IN INDIAN COUNTRY — KEYNOTE ADDRESS

James W. Hampton, M.D.

The problem of cancer in Indian people is a recent one (1,2). Currier wrote in 1891 that "malignant diseases of the reproductive organs seem almost unknown among Indian women" (3). Cancer was called "kachombi" by the Choctaws. My grandmother, who was born in 1865 in the Choctaw Nation in Indian Territory, believed that Indians never had cancer. Cancer was a "white man's disease" (4). At the beginning of the twentieth century the epidemiological statistics for cancer were crude at best for all Americans, and specific anthropological and medical literature cited that cancer was "rare" in this special population of Native Americans (3,5,6). Today, the existence of cancer in Indian Country has still not been recognized as a problem by Indian health care deliverers, clinical investigators, private cancer societies, foundations, or even by Native Americans themselves (2).

Clearly cancer has increased throughout this nation and among the Indian population during the twentieth century. For example, from a "rare" disease at the beginning of the century, the incidence of some forms of cancer in the Northern Plains American Indians and in Alaska Natives has been documented to exceed that of the "white" population (7). The SEER data indicates that there is a lower incidence of cancer among southwestern American Indian tribes than in the general population, but it also indicates that there is a higher mortality rate from cancer than is found in other special populations (8). This high mortality rate may be attributed to a more virulent disease or to stage-to-stage delayed diagnosis of cancers that might have been put into remission by "state-of-the-art" treatment. Lung cancer, which figures prominently in the Northern Plains and Alaska, was significant in the southwestern Indians only if they were employed as uranium miners (9,10).

Sievers stated in 1983 that the risk of cancer was significantly lower among the American Indians of North America than in the general population, but that this lower rate was largely confined to men (11). He did remark that uterine cervical cancer as well as gallbladder cancer were the exceptions. He attributed the low risk of lung cancer in the southwestern tribes to the uncommon practice of very little cigarette smoking in that group. He further remarked that the heavy cigarette smoking in Alaska Natives accounted for their striking increase in mortality due to cancer of the lung.

Carcinoma of the uterine cervix in American Indian

women is twice that for U.S. women, all races. The SEER data showed that 80% of the deaths among southwestern American Indian women occurred in patients over 40 years of age. Late detection was implicated as a cause of the high mortality rates in these women who are beyond the child-bearing period (12,13).

The high risk of Alaskan Native women for carcinoma of the uterine cervix has been described by Lanier and her co-workers (13). The authors also describe disproportionately high rates of hepatocellular carcinoma linked to hepatitis B and nasopharyngeal carcinoma linked to the Epstein-Barr virus. Lanier and Knutson (7) reviewed the Alaska Native statistics for the period 1969-1983 and have expressed concern over the increase in cancer in this special population. Much of this increase was due to lung cancer, which was attributed to high rates of cigarette smoking.

Accurate rates for cancer in Indian peoples are often difficult to calculate (14). For example, cancer statistics in the Oklahoma area (Oklahoma-Kansas), California area, and Portland area (Washington-Oregon and Idaho) are inaccurate because of problems with accurate reporting of Native American race on death certificates and therefore must be excluded from national statistics although these areas have large populations of American Indian people.

The medical community is beginning to become aware of the problems of cancer in this special population. In April 1987, the First Biennial Symposium on Minorities and Cancer was held in Houston, Texas, sponsored by the M. D. Anderson Regional Cancer Center. Dr. Lovell Jones, who chaired that meeting, requested that the Association of American Indian Physicians propose a speaker for the topic of "Cancer in American Indian/Alaska Natives." As the past-President of the Association and a medical oncologist, I was asked to make this presentation (8).

The lecture was published, and the SEER data which described the "lower" incidence of cancer in American Indians was acknowledged. However, we pointed out that the southwestern Indians had long been observed to "lack" cancer as a medical problem, but they demonstrated unusually high rates of certain cancers such as those of the gallbladder and the biliary tree (15). Gastric cancer and pancreatic cancer were also prominent in American Indians, and the mortality rates for these cancers were not declining as they were in the Caucasian



population (18). Genetic factors were suggested to predispose the American Indian population to gallbladder and biliary tract cancer (17). Fraumeni had described biliary tract cancer as extraordinarily high in all indigenous peoples of the Western Hemisphere (15).

Boss et al. (18) found that gallbladder cancer in Alaska Native men and women developed nearly as often as it occurs in southwestern American Indians. They remarked about the cultural dissimilarity between the rates for this cancer in Alaska Indians and in the Eskimo-Aleuts, which might further suggest a genetic factor. Morris and co-workers had previously shown the comparable incidence of gallbladder cancer in Hispanics of Indian ancestry (17). Valway in 1992 reported the age adjusted mortality rates from gallbladder and biliary tract cancer in American Indian women to be significantly higher than the U.S. rates for these cancers (14). For example, the Tucson area had a rate for this cancer 24 times the U.S. all races rate.

## THE NETWORK FOR CANCER PREVENTION AND CONTROL

As a consequence of our observations that American Indians/Alaska Natives have certain similarities but do not represent a homogeneous population, particularly in regard to incidence of specific cancers, a meeting was held in 1987 in Washington, D.C., and an interagency agreement was established between the National Cancer Institute and the Indian Health Service. In this landmark meeting, two federal agencies were brought together to discuss a growing problem in this special population.

Through the efforts of Dr. Claudia Baquet, then Chief of the Special Population Branch, Division of Cancer Prevention and Control of the National Cancer Institute, a Network for Cancer Control Research in American Indians/Alaska Natives was formally established in April 1990, the same Network which is responsible for holding this conference. The mission statement of the Network acknowledged the increased incidence and mortality from cancer among American Indian and Alaska Natives in the twentieth century and committed its efforts to restore the health of this population.

The Network in 1990 identified its long-range goals, objectives, and tasks. Subcommittees were selected to alter the racial misclassification of American Indian/Alaska Natives. Underreporting of cancer in urban populations was found. The Network identified the need to educate researchers and providers about this growing public health problem and sought advocacy for funding of health care and research on cancer in Native Americans.

In 1991 a grant application was submitted by the Network's members to the National Cancer Institute to hold a conference on the subject of "Cancer in American

Indians/Alaska Natives." Members of the Network, with Dr. Jennie Joe as Principal Investigator, were awarded a conference grant through the Native American Research and Training Center of the University of Arizona. A "Cancer Education Program Survey" was also developed for health care deliverers.

In 1992 the Network developed a "National Strategic Plan for Cancer Prevention and Control to Benefit the Overall Health of American Indians and Alaska Natives." They presented this plan in October 1992 to Dr. Samuel Broder, Director of the National Cancer Institute and Dr. Everett Rhoades, Director of the Indian Health Service on the occasion of the Celebration of Survival for Five Hundred Years. The purpose of this plan is to enhance the awareness in federal agencies and in others about the problems of cancer among American Indian and Alaska Native populations. It includes an executive summary, introduction, overview of the Plan's issues and recommendations for federal agencies, as well as action items and outcome measures for each recommendation.

## SUMMARY AND RECOMMENDATIONS

To obtain correct cancer statistics in American Indians, there must be a collaboration between SEER sites, state tumor registries, and the Indian Health Service. Previous SEER data are based on southwestern American Indians only who have "markedly different cancer mortality patterns from other Native Americans" (14). Furthermore, the gene pool of indigenous people, especially women of the entire Western Hemisphere, needs to be explored and mapped to look for an etiological source for the origin of gallbladder and biliary tract cancer.

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*(continued on page 272)*

# EPIDEMIOLOGY OF CANCER IN ALASKA NATIVES

Anne P. Lanier, M.D., M.P.H.<sup>(1)</sup>

## ABSTRACT

In contrast to many other Native American groups in the United States, cancer incidence data are available for the Alaska Native population from 1969 to present. These data support the fact that cancer patterns in this population differ from all other U.S. populations and are most similar to Eskimos (Inuit) of other circumpolar countries. The unique cancer patterns and the distribution of the population far from sophisticated medical centers strongly impact the cost/benefit ratios of recommended cancer screening procedures. Cost/benefits of cancer screening and data showing that more than one-third of cancer cases and cancer deaths are tobacco related, necessitate that cancer control efforts in this population should focus on eradication of tobacco.

## INTRODUCTION

Cancer incidence patterns in Alaska Natives (Eskimos, Indians, Aleuts) differ from those of U.S. Whites and from those of American Indians (1-5). Although cancer was once thought to be rare in the Alaska Native population, current total cancer incidence rates are similar to those of the U.S. when adjusted for the age distribution of the population. In comparison with the general U.S. population, excess cancers occur in Alaska Natives in the following organ sites: nasopharynx, salivary gland, esophagus, stomach, liver, gallbladder, cervix, and kidney. On the other hand, relative deficits have been found for the following cancers: prostate, breast, uterus, bladder, and leukemia/lymphoma.

Cancer mortality rates are higher in the Alaska Area Native Health Service than in any other population served by the Indian Health Service (6-7). The excesses in mortality are even greater than the excesses in incidence rates. Thus survival rates are low, perhaps due to late stage at diagnosis, less than optimal treatment, or poor outcome due to other factors.

Many factors combine to make cancer prevention and control a particularly great challenge among the

Alaska Native population. These include: differences in cancer patterns; cultural and language barriers; unique diet, food sources, and preparation; distribution of the population in small, remote communities scattered over a very large geographic area, etc. Although excellent primary care for acute disorders are provided in the communities by a unique health aide program, it is not possible to offer expensive, highly technical and sophisticated medical procedures in the majority of communities. Transportation costs alone to these services may exceed the costs of services.

This article summarizes current knowledge of the epidemiology of cancer among Alaska Natives. These data are presented to focus cancer control efforts toward activities which will be most cost effective and have the greatest impact on health in both the near and long term future.

## METHODS

In 1974 efforts were initiated to accurately describe cancer incidence and mortality among Alaska Natives. Since that time efforts have continued to identify all Alaska Natives diagnosed with invasive cancer who were residents of Alaska at the time of diagnosis. The term Alaska Native is used to include Indians, Aleuts, or Eskimos whose family origins are Alaskan. The population of Alaska Natives based on the 1990 census totalled 84,700, up from 50,900 in 1970. Alaska Natives who are eligible to receive health care from the Indian Health Service are registered. The registry is currently housed at the Alaska Native Medical Center (ANMC), but includes all Natives diagnosed with cancer, not just those treated at ANMC. Registration methodology has been described in previous reports (1-5). Essentially all possible sources are reviewed to identify potential cases: in- and outpatient diagnostic indices, tumor registries, pathology reports, death certificates, etc. Data collected have followed National Cancer Institute's SEER program. From 1969 to 1983 limited data items were collected that focused on describing incidence patterns. Since 1984, an attempt has been made to include all data items collected by SEER programs as well as additional demographic and risk factor data of importance to this population.

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The data compiled for this paper were felt to be complete for the years 1969-83 and have been previously summarized and reported. Data for the years 1984-88 are preliminary.

## RESULTS

Over 2100 invasive cancers were identified and registered among Alaska Natives for the time periods 1969 to 1988. Although a few cases are based on death certificates only, over 96% of the cases registered in the Alaska Native Cancer tumor registry are confirmed by histology or cytology. In the 1980s the number of invasive cancer cases per year averaged 150. The most frequent cancers diagnosed in men and women separately and combined are shown in Table 1. In men, three cancers (lung, colon/rectum, prostate) account for half of all the cancers diagnosed, while in women two of the same (lung, colon/rectum) plus breast cancer are responsible for half of all cancers diagnosed.

**Table 1**

**Cancer Incidence  
Rank Order  
Alaska Natives**

MEN	WOMEN	TOTAL
lung	colon/rectum	lung
colon/rectum	breast	colon/rectum
prostate	lung	breast
stomach	cervix	kidney
kidney	kidney	stomach
nasopharynx	ovary	prostate
lymphoma	pancreas	cervix
leukemia	stomach	pancreas
testes	thyroid	gallbladder
liver	gallbladder	nasopharynx

The age-specific incidence curve for cancer in the Alaska Native population follows that of the U.S., i.e., there is a rapid increase in cancer risk beginning at about age forty. Based on National Census data, the Alaska Native population is characterized by a large number of young persons (about 50% under age 20) and a small proportion of elders (5% over age 65). Because of this, clinicians are often impressed by the relatively large number of young cancer patients they treat. However, because of the marked increase in risk of cancer with age, over half of the patients diagnosed with cancer even in the relatively young Alaska Native population are diagnosed at age 50 or older.

The most frequent causes of cancer death based on data for 1984-88 are shown in Table 2. Cancer site is much less frequently specified and/or correct on death certificate than data available on incidence cases in the

registry. However, lung cancer is the leading cause of cancer death in both Alaska Native men and women, accounting for over 30% of cancer deaths.

**Table 2**

**Cancer Deaths  
Rank Order  
Alaska Natives**

MEN	WOMEN	TOTAL
lung	lung	lung
colon/rectum	colon/rectum	colon/rectum
stomach	breast	stomach
pancreas	cervix	pancreas
esophagus	pancreas	breast

The distribution of cancers by IHS service unit was analyzed by comparing the proportion of the Native population residents of the service units with the proportion of total cancers diagnosed among Native residents of that service unit. The distribution of all invasive cancers by service unit is similar to that of the Native population; no service unit appears to have a markedly disproportionate number of cancer patients. Analysis of the distribution of each cancer site is hampered by the small number of cases. However, only cancers of the liver and esophagus appear to be clustered geographically, which may be confounded by genetic, cultural and familial distribution. For example, for cancers of the nasopharynx and liver, roughly one third of the patients diagnosed with these cancers are members of multi-case families.

When data for 1969 through the present are analyzed for trends, the most dramatic increase in cancer occurred for cancer of the lung. The increase in lung cancer alone accounted for nearly all of the increase in the overall cancer rates. Cancer mortality data show a similar trend, i.e., a dramatic rise in lung cancer mortality in both men and women with the increase occurring earlier in men than women. On the other hand, cancers which occur at unusually high rates — nasopharynx, liver, gallbladder, etc., do not appear to be decreasing in incidence.

Because of funding difficulties statewide, comprehensive Native registry efforts were discontinued in 1985, then renewed in 1989. Data on staging, treatment, and follow-up have only been included in the registry information since its revival in 1989 with support from the National Cancer Institute. In 1989, efforts were focused on identifying all cases from 1984 to present and including information on staging, treatment, and follow-up. Preliminary review of the 1984-88 data indicates a low overall survival rate (30%) for invasive cancers, with only approximately one-third of the cases diagnosed when the stage of the cancer is localized to the site of origin.

## DISCUSSION

Although once thought to be rare or even nonexistent, cancer is now a major problem among Alaska Natives, ranking as the third leading cause of inpatient days caused by illness. Among causes of death in the Native population, cancer vies for second place, markedly exceeded only by intentional and unintentional injuries. Among Alaska Native women, cancer is now THE LEADING cause of death.

Cancer patterns in the Alaska Native population are unique and differ from those of other comparison groups — Alaskan non-natives, U.S. whites, U.S. minority populations, American Indians in the contiguous United States, and other Asian races in the U.S. However, cancer patterns in Alaska Natives are quite similar to those of other Eskimo (Inuit) populations in Canada and Greenland (8). Unfortunately cancer sites commonly considered “Western” (lung, colon/rectum, breast, prostate) already occur in relatively high numbers or are increasing, while those unusually common in this population (nasopharynx, gallbladder, kidney, liver) show little evidence of decreasing in incidence. Comparison of the epidemiology of cancer in this population, along with special studies here and among populations with differing patterns, holds clues to cancer etiology (9).

From the perspective of cancer prevention, the data clearly indicate that at least ONE THIRD of new cancers and cancer deaths are TOBACCO related. Clearly eradication of tobacco use from this population will have the most profound effect on cancer incidence and mortality and, of course, result in marked improvements in morbidity and mortality from nearly every other major cause of death and disability (cardiovascular and cerebral vascular disease, chronic bronchitis and emphysema, pregnancy outcome, SIDS, respiratory disease in children and adults, etc.).

From a perspective of cancer control, it should be noted that two cancers for which there are proven effective screening techniques (breast and cervical cancer) are among the leading causes of cancer. Although screening does not alter incidence, screening should impact mortality. However, although Pap smears have been available at no cost to the Alaska Native population for many years, current mortality rates from cervical cancer still exceed by several fold those of the U.S. population. The reasons for this are under investigation. On the other hand, screening for breast cancer, if it is to include mammography, is not something that has been or can be readily available to the Native women in the predominantly rural, remotely scattered communities. Costs of screening the entire population of native women according to current national recommendations have been estimated (9,10) and require a serious look at cost/benefits. It is exceedingly difficult and costly to offer

cancer screening services as currently recommended for the nation. Innovative approaches are therefore needed for both prevention and control of cancer in this population.

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# National Cancer Institute's Native American Cancer Research Projects

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## ABSTRACT

The purpose of this paper is to briefly describe several of the NCI Native American initiatives that illustrate the diversity and breadth of NCI's commitment to indigenous peoples. The National Cancer Institute (NCI) began introducing Native American cancer initiatives in 1987. Initiatives which are described in the paper include the following: four intervention research projects on cervical and breast cancer among Native Americans; four intervention research projects on the prevention of tobacco and dietary cancer risk factors among American Indians; investigator-initiated developmental research grants on cervical and breast cancer; intra-agency agreements with the Indian Health Service; the Network for Cancer Control Research among American Indian and Alaska Native Populations; Native American Training Opportunities Meeting; Data-based Intervention Research projects which focus on Native Americans.

## INTRODUCTION

The purpose of this paper is to briefly describe several of the NCI Native American initiatives that illustrate the diversity and breadth of NCI's commitment to indigenous peoples. The National Cancer Institute (NCI) began introducing Native American cancer initiatives in 1987.

The National Cancer Institute (NCI) is the Federal Government's principal agency for conducting and supporting research on cancer and is responsible for overseeing implementation of the National Cancer Act which coordinates a national research effort on cancer cause, prevention, detection, diagnosis, treatment, rehabilitation, and control. The Division of Cancer Prevention and Control, Special Populations Studies Branch (SPSB) of NCI, is designed to address NCI's year 2000 goals to reduce and eliminate the differentials in cancer inci-

dence, mortality and survival between minority/special populations and the general population. The terms "special populations" includes Alaska Natives and American Indians among other populations who are known to experience high cancer rates.

## Cooperative Agreements

The Special Population Studies Branch, CCSP, DCPC, NCI released two requests for applications (RFA) in 1989 focusing on intervention research in Native American populations: the first was "Avoidable Mortality from Cancers in Native American Populations" (cervical and breast cancer), and the second was "Primary Prevention of Cancer in Native American Populations" (tobacco use and diet). Eight projects were awarded during Spring and Summer 1990 as five year cooperative agreements with the NCI. Four projects were for research on avoidable mortality and four were for research on primary prevention (see Table 1).

## AVOIDABLE MORTALITY FROM CANCERS IN NATIVE AMERICAN POPULATIONS

The goal of NCI's "Avoidable Mortality from Cancers in Native American Populations" Program was to identify key factors that contribute to avoidable mortality from specific cancers such as cervical and breast cancer; to develop and evaluate the effectiveness of community baseline interventions; and to reduce mortality from specific cancers (e.g., cervical, breast). The four projects focused on Native American communities: one targeted American Indians in the Southeast; one focused on urban Indians in seven geographically diverse settings; one targeted rural Native Hawaiians; and one focused on Alaska Natives in an urban setting and a rural setting. Knowledge, attitudes, and screening practices were addressed. All were random trials, and all involved interventions.

### 1. Bowman Gray North Carolina Project

The "Prevention of Cervical Cancer in Native American Women" in North Carolina focused on women from the Lumbee Tribe and the Eastern Band Cherokee Tribe.

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**Table 1****Study Components of Avoidable Mortality from Cancer in Native American Populations Projects in Alaska, Minnesota, North Carolina, and Hawaii.**

Study Components	Alaska	Minnesota	North Carolina	Hawaii
Target Population	Alaska Native Women, Age $\geq 18$	Native American Women Age 18+	Native American Women Age 18+	Native Hawaiian Women Age 19+
Subjects	Stratified random sample from IHS medical service recipients	First 160 patients from each clinic	Random samples from Lumbee tribal rolls; area sample of Cherokee reservation	Random generated telephone numbers in selected census tracts
Sample Size	500 from Anchorage, 125 from St. Paul	160 patients from each clinic	1000 each from Lumbee and Cherokee tribes	1200 baseline control, 1400 full-scale intervention
Sites	Urban and rural communities in Alaska, Anchorage and St. Paul Island	7 sites, 3 matched pairs plus one addition	Lumbee and Cherokee populations. Each population managed as separate experiment	Experimental group selected from Wai'anae Coast. Control selected from Native Hawaiian population on Oahu

The Cherokee are a reservation tribe residing in the western-most region of North Carolina. Women from each tribe were randomly divided into intervention and control groups after recruitment.

The purpose of the project was to prevent cervical cancer by increasing the proportion of women who received regular Pap smears and returned for follow-up care when necessary. Women aged 18 and older who were enrolled members of the Cherokee and Lumbee tribes were eligible for inclusion in the project. The Cherokee study population included all women age 18 and older residing on the reservation in western North Carolina. The Lumbee study population was randomly selected from female tribe members who were residents of Robeson County, North Carolina (the traditional homeland of the tribe).

Project goals were to be achieved by development and implementation of a focused, culturally sensitive, community health education intervention. Supported by extensive community analysis, the intervention was presented to women individually by trained Native American lay health educators. The intervention included an initial presentation of information about cervical cancer and its prevention. Information presented was reinforced and augmented in subsequent contact with participants through mail and telephone.

A total of 1000 eligible women were recruited in each population (2000 total). These women were randomly assigned to one of four groups of 250. Five hundred of the women in each population (two groups) received the pretest interview, and one of each group of

two received the intervention. One of the remaining two groups that were not interviewed also received the intervention. This Solomon Four Group design was selected because it allowed estimation of the effect of the pretest interview as an intervention. All women are to be interviewed after the intervention. Outcome measures include changes in knowledge, attitudes, and behaviors with regard to cervical cancer and early detection. Data on Pap smears were also collected from medical providers servicing each population. These data were to be used to evaluate the accuracy of responses on the pretest interviews.

## 2. The American Indian Health Care Association Project

The "Urban Native American Women's Cancer Prevention Project" focuses on Native American women age 18 and over from seven urban areas at seven urban Indian Health Centers and from a clinic-based patient outreach to urban Native American women in four intervention communities. The project sites include clinics and Indian Health Boards in Seattle, Salt Lake City, Oklahoma City, Tulsa, Detroit, Milwaukee, St. Paul, and Minneapolis. The purpose of the project is to: 1) assess cancer prevention knowledge, attitudes, and behaviors; 2) develop culturally-sensitive prevention intervention strategies; and 3) evaluate the effectiveness and efficacy of those strategies.

Both primary and secondary prevention intervention will be provided through four well-established urban



Native American health programs; three other matched clinic settings will serve as comparison sites. The intervention will consist of 1) a review of patient records to determine the proportion of eligible women who are receiving annual Pap smear examinations and subsequent contact of eligible non-screened clients by field workers to induce their participation; 2) educational outreach into the community by trained Indian health community health workers for primary prevention; 3) cancer education programs provided by health professionals for women who come into the clinic for screening; 4) health provider training for Indian nurse educators on clinical cancer prevention, and for non-Indian community health organizations on providing culturally sensitive treatment to urban Indian women; and 5) case management services for women patients to assess their options for health care services and reduce financial barriers to follow-up care.

Evaluation of the study will be accomplished by measuring the number of participants who are screened as well as those who receive follow-up treatment.

### **3. The Wai'anae Coast Cancer Control Project**

The "The Wai'anae Coast Cancer Control Project" targets Native Hawaiian women 18 years of age and older. The purpose of the project is to test the effectiveness of an integrated, community-driven cancer control intervention to increase breast and cervical cancer screening rates as well as to increase knowledge, to change attitudes, and to improve behavior (KAB) scores among participants. The primary intervention is the development of health-activated mutual support groups ("Kokua Groups") within existing Hawaiian social networks.

This project will test the effectiveness of an integrated, community-driven cancer control intervention that has been designed to take advantage of Native Hawaiian social and family networks and their sense of "Kokua," a Hawaiian social concept that encourages mutual support of community members. "Kokua" groups will be formed to provide linkage between health workers and native Hawaiian women and to encourage mutual-support in addressing cancer issues among community members. Project effectiveness will be measured by changes in mammography and PAP screening rates and in increased knowledge, attitudes, and practices (KAP) scores among Kokua group members (n=1400). Process evaluation will determine project components that were influential in affecting screening rates. Community diffusion will be assessed by telephone surveys of an empaneled random sample of community members (n=600). KAP changes among this telephone survey sample will be compared to a telephone survey of Native Hawaiian women outside the intervention area (n=600 empaneled). An additional group of 600 women

in both populations will be surveyed to control for the "learning" effect of participating in telephone surveys

### **4. Alaska Area Native Health Service and Aleutian/Pribilof Islands Association, Inc.**

The "Prevention of Cervical Cancer in Alaska Native Women" project targets Alaska Native women age 18 and over in two locations: an urban area, i.e., Anchorage, Alaska, at the Alaska Native Medical Center, and one rural area, St. Paul Island, in the Pribilof Islands. In Anchorage, 500 women will be enrolled from a random sample of the 5000 eligible Native residents of the area. In St. Paul, all adult women (approximately 125) are eligible to participate.

The long-term objectives of this project are to reduce the morbidity and mortality from invasive cervical cancer in Alaska Native women. The specific aims are: to promote knowledge and awareness of this disease, its risk factors, and appropriate screening programs; and to enhance the existing cervical cancer screening services and follow-up care of precancerous lesions.

Participants agree to complete face-to-face interviews, pre- and post-test intervention, focusing on their knowledge, attitudes, and behaviors with regard to cancer in general and cervical cancer in particular. Review of medical charts and records from cytopathology labs provide documentation of actual pap smear screening activity.

Intervention strategies will be implemented following enrollment in the project. One intervention is a special demonstration women's health clinic. This Women's clinic includes: extended evening hours, hour-long appointments, staffing by women providers and nurse practitioners, comprehensive health surveillance for women of all ages, pap tracking services, individual patient education, mammography services, and tobacco cessation classes.

Outcome measures include: annual screening rates for women in the project compared with non-participants, rates of completion of scheduled appointments, and the time interval from the receipt of abnormal pap smear results to the initiation and completion of follow-up treatment.

## **PRIMARY PREVENTION OF CANCER IN NATIVE AMERICAN POPULATIONS**

The goal of this initiative is to develop innovative tobacco and/or dietary intervention programs and to determine the long-term effectiveness of these programs on the prevention of cancer among Native Americans. These projects are developing a variety of techniques to prevent or reduce habitual tobacco use (NOTE: the projects are distinguishing between Native American ceremonial tobacco use and habitual use). Two projects

target school-age American Indian children and stress dietary modification and prevention of habitual tobacco use. Another project targets American Indian adult tobacco cessation, and the fourth is working with tribal councils on developing tobacco use policies. The settings include urban clinics, rural settings, and reservations. Knowledge, attitudes, and practices are also being addressed. All four of the funded projects focus on American Indians (See Table 2).

## COLLABORATIVE EFFORTS AMONG COOPERATIVE AGREEMENTS

Collaboration exists in multiple tasks among the grantees. The Avoidable Mortality research teams collaborate on tasks to increase the type of data collected on selected knowledge, attitudes, and behavior cervical cancer survey items. The Primary Prevention grantees adopted the survey instrument developed for use from one project and implemented it in each research site. In addition, the Avoidable Mortality and Primary Prevention grantees have collaborated on joint tasks such as participating on a survey of principal investigators to document the process and procedures utilized to effectively work with Native communities. The Cooperative Agreement grantees are collaborating on approximately three joint publications.

## R01 INVESTIGATOR-INITIATED NATIVE AMERICAN RESEARCH PROJECTS 1989-1992

The Special Populations Studies Branch released a request for applications (RFA) in 1989 for Native American developmental research. In response to that RFA, two applications were funded.

### Cervical Cancer Among American Indian Women

Farmer's "Cervical Cancer Among American Indian Women" was a three year project implemented in Southern California. The California State University Long Beach and the American Indian Free Clinic, located in Los Angeles County, collaborated to develop and administer a culturally sensitive cervical cancer knowledge, attitudes, and behavior instrument to Indian women residing in the greater Los Angeles-Orange County area. The project aims were to (1) assess the need for cervical cancer control interventions; (2) determine the barriers to cancer control programs; and (3) develop a culturally sensitive instrument for data collection. This instrument was used to determine the prevalence of cervical cancer symptoms; determine the prevalence of risk factors; measure, knowledge, attitudes, and practices; and assess accessibility and acceptability of cervical cancer screening and treatment. The total sample size was 1500 American Indian women; 800 American Indian women participated in the pilot test and 700 in the final survey.

### The Sioux Cancer Study

The purpose of Welty's "Sioux Cancer Study" was to assess cancer mortality and cancer risk factors among

**Figure 2**  
**Study Components of Primary Prevention from Cancer in Native American Populations Projects in Oregon, California, New Mexico, and New York.**

Study Components	Oregon	California	New Mexico	New York
Target Population	39 Tribes in OR, ID and WA	American Indian Adults	5th and 7th grade American Indian students	American Indian adolescents
Subjects	Random assignment of tribes to early or late intervention	Random assignment of 14 rural and 4 urban Indian Clinics	Random assignment of classrooms to 1 of 3 interventions	Random assignment of after-school programs to 1 of 3 interventions
Sample Size	39 Tribes	1400 clinic users	500 Native children	Approximately 250 Native American Adolescents
Sites	Tribes located in Idaho, Oregon, and Washington State	Indian Clinics located in Northern California	Alamo, Santa Fe, Crownpoint, San Juan Laguna-Acoma, Thoreau, Borrego Pass, Espanola, Smith Lake (NM); Aneth, UT, Hopi and Keams Canyon (AZ)	Native programs located in Maine, New York, Connecticut, New Jersey, and Rhode Island



1,521 Sioux Indians aged 45-74 who were members of the Devil's Lake, Cheyenne River, and Oglala Sioux Tribes. The study also determined the adequacy of screening for cervical, uterine, and breast cancer and the acceptability of screening programs in a high risk age group of Sioux women.

During this study, three instruments were designed, pilot tested, and utilized. The knowledge, attitudes, and beliefs questionnaire for cervical and breast cancer (Mammography Survey I) was administered to all women who were participants of the Sioux Cancer Study and who came to receive mammography screening as part of this study. Through the grant, screening mammography was provided to 559 women in their target communities. In communities where mammography screening was provided through the mobile unit, the KAB survey was also administered to women who did not come in to receive the mammography screening. The responses of participants and non-participants to the Mobile Screening Program were compared. The patient satisfaction questionnaire (Questionnaire for Mammography Participants) was administered to all women after they had received mammography screening and was helpful in assessing the response of Sioux women to screening. Women who did not return for the screening mammogram were contacted and, if they consented, completed the Mammography Survey II (a non-participant questionnaire) to determine why they did not participate in the screening.

## **DOCUMENTATION OF THE CANCER RESEARCH NEEDS OF AMERICAN INDIANS AND ALASKA NATIVES**

The **purpose** of this project was to formulate a series of publications which provide a resource and reference to assist in the formulation of culturally acceptable cancer prevention and control research projects or programs. The initial monograph, *Documentation of the Cancer Research Needs of American Indians and Alaska Natives*, is a *brief* overview of the cancer problem among American Indian and Alaska Native People living in urban, rural, reservation and village sites. It is not designed to be read from cover to cover, but rather that the reader will utilize appropriate sections that may assist in the formulation of a cancer prevention and control program for one's local Native community. This document is neither all encompassing nor comprehensive, but is rather an introduction to the cancer problem as it currently exists among American Indians and Alaska Natives. Because specific tribal information is rarely available, reservations, urban Indian clinics, and other tribal settings are encouraged to collect and record cancer data for their community.

## **Limitations of this Publication**

These monographs represent a series of proposed publications that are designed to be of assistance to program planners, researchers, and tribal councils and boards. It is not possible to include everything one needs to know about the research process in one monograph. For your information, the suggested monographs include the following:

- Monograph #1: Documentation of the Cancer Research Needs of American Indians and Alaska Natives
- Monograph #2: Documentation of the Cancer Research Needs of Native Hawaiians and American Samoans
- Monograph #3: Cancer Risk Factors among Native Americans
- Monograph #4: Planning Cancer Prevention Research Projects among Native American Populations
- Monograph #5: Initiating Local Native Support for Cancer Prevention and Control Programs
- Monograph #6: Participation with and recruitment of Native Americans in Clinical Trials and other types of Research Projects

## **CANCER PREVENTION AND CONTROL WORKSHOP FOR AMERICAN INDIAN AND ALASKA NATIVE POPULATIONS**

The National Cancer Institute's *Cancer Prevention and Control Workshop for American Indian and Alaska Native Populations* was a two-day workshop held in Tucson, Arizona, on March 23-24, 1990. The purpose of the workshop was to assist the participants in translating cancer data into culturally sensitive terminology and concepts appropriate for their local tribal community. The two-day workshop included 45 participants from diverse regions of the U.S. Over two-thirds of the participants were enrolled tribal members and over 80% were female. The participants included health care providers, clinicians, and indigenous aids (e.g., Community Health Representatives) from American Indian and Alaska Native reservations, urban clinics, tribal hospitals, and Indian Health Service (IHS) sponsored health care settings. This workshop was one component of the Special Populations Studies Branch's efforts to implement research capacity development for the American Indians and Native Alaskans in addressing their cancer issues.

The agenda included the following topics: cancer "statistics" and "Native Peoples," high risk behaviors,

cancer prevention screening and treatment among Native populations, traditional medicine and cancer, cultural barriers related to cancer prevention, implementing cancer prevention and control in different settings, making educational messages culturally sensitive, small group work with educational materials, making the message understandable and culturally sensitive, making the aid understandable (visual, video, audio), simple messages with audio tapes, small group work with simplifying messages, and examples of culturally sensitive video-tape. The cancer sites which were highlighted in the workshop were cervical, breast and lung cancer.

Several of the participants of the workshop have since been hired as consultants to assist with developing, concept-testing and/or pretesting cancer education materials designed for Native people.

### **INTRA-AGENCY AGREEMENT WITH INDIAN HEALTH SERVICE - ALASKA NATIVE AREA - FIVE-YEAR CANCER SURVEILLANCE OF ALASKAN NATIVES (February 1989 to January 1994)**

The purposes/goals of this agreement were: (1) to maintain active cancer surveillance and document all newly diagnosed Alaska Native patients with invasive cancer since 1969 and in situ (CIN III) cancers starting in 1990; and (2) to collect and computerize detailed information on all cancer patients including demographics, basis of diagnosis, stage, treatment, and follow-up. This IAA allowed for tasks, including but not limited to the following: provide follow-up on cancer survival of diagnosed cancer patients as well as implement an Alaska Native surveillance system, providing sufficient detail so that each cancer is classified and coded in accordance with written procedures. Additional data items include the following:

- Ethnicity to linguistic subgroup
- Quantum native blood
- Village of birth
- Village of diagnosis
- Use of tobacco products
- Alcohol usage history
- Information on whether cancer was detected by screening

In addition, the Alaska Area Office of the Indian Health Service is utilizing all available methodologies to obtain active follow-up on all cases diagnosed in 1969 forward among Alaskan Native residents of Alaska. They are performing basic data edits, updating patient and tumor specific information, and facilitating preparation of the SEER submission tapes. They are also cooperating with the NCI field staff in its quality control activities.

### **INTRA-AGENCY AGREEMENT (IAA) WITH INDIAN HEALTH SERVICE - TUCSON RESEARCH OFFICE (July 1989 to June 1993)**

This IAA allowed IHS to work collaboratively with NCI in research projects to determine how and why cancer survival is so poor among Native Peoples as well as to determine the risk factors which are responsible for the rapid rise in cancer among Native Peoples during the last thirty years.

This IAA was designed to encourage consortia of existing cancer control-related research institutions and relevant regional health agencies to pool their resources in an administrative mechanism that would effectively address cancer control research projects directed towards achieving the year 2000 cancer goals. This Agreement directly supported NCI objectives by (1) providing the impetus for a specialized cancer center consortium, and (2) developing program activities which enhance the goals for cancer prevention and control for special populations at NCI.

This Intra-Agency Agreement between the Indian Health Service (IHS) and the National Cancer Institute (NCI) was intended to provide support for the planning and development of research projects directly related to primary prevention and avoidable mortality. The ultimate goal of this agreement was to bring together the expertise and objectives of the NCI with the service responsibilities of the National IHS. It was further anticipated that this IAA will support the development of specific projects that will provide the experience and data base necessary for the National IHS to eventually apply for a consortium cancer center planning grant.

### **Examples of Accomplishments from the 1989-1992 NCI-IHS Intra-agency Agreement**

Nine research projects were completed as part of this intra-agency agreement. The IHS publication "Cancer Incidence in American Indians and Alaska Natives" in the *American Journal of Public Health* is an example of one project (scheduled for release in 1993). Another project focused on mortality databases to determine racial misclassification. The New Mexico SEER Registry at the time of database review, contained few errors (only 0.13 percent). However, racial misclassification ranged from 20-40% for the Fred Hutchinson Cancer Research Center and Montana Central Tumor Registry. They also found underreporting of Indian race on state death certificates in California, Oklahoma, and Washington (the error rates are likely higher in California and Oklahoma, which according to the 1990 Census are the two states with the highest number of American Indians). The IHS areas which were found to NOT have significant problems with racial misclassification at



death included Alaska, Albuquerque, Phoenix, Bemidji, and Aberdeen.

The IHS developed, pretested, and administered the "IHS Providers' Knowledge-Attitudes-Beliefs-Behaviors (KABB)" survey to 1050 IHS providers (response rate was 70 percent, n=739). Among the findings was that clinicians in six IHS administrative areas reported IHS policies as an important barrier to screening mammography (diagnostic mammograms are allowed, but not screening mammograms) on their patients and most IHS personnel did not understand the importance of clinical breast examinations. As a result, provider education is being planned.

## **DATA-BASED INTERVENTION RESEARCH PROJECTS WHICH ARE FOCUSED ON AMERICAN INDIANS**

The goal of the Data-based Intervention Research (DBIR) Program is to work with state and local health departments to build the foundation for on-going programs that will translate cancer prevention and treatment science into practice across the United States. Currently, 21 states and the District of Columbia are supported by the DBIR Program. Initially, six states received awards. Eight additional awards were made in 1989, and eight more states received funding in 1990 under the DBIR Program.

Under these awards, recipients analyze available data to develop state cancer control programs and begin the implementation and evaluation of high priority cancer control interventions that meet the unique needs of each state. Among the major advantages for participants in the DBIR program are opportunities to share and benefit from the experiences of other states that are also developing and implementing cancer control programs.

Three of the twenty-two projects include American Indians as the target population in Nebraska, North Dakota, and Alaska. The Nebraska DBIR is for cervical cancer detection and the North Dakota DBIR is for tobacco use reduction, breast cancer detection, and cervical cancer detection. In 1992 the Alaska project began to develop a state cancer control plan that will include information about cancer and Alaska Natives and will serve as the basis for targeting cancer control interventions.

## **MINNESOTA STATE DEPARTMENT OF HEALTH AMERICAN INDIAN CERVICAL CANCER SCREENING RESEARCH PROJECT**

The purpose of the Minnesota Department of Health "Increasing Cancer Screening among Underserved Women" (1989-1995) is to design and evaluate two

approaches to increase breast and cervical cancer screening rates among women who are predominantly indigent, elderly, and/or Native American, 40 years and older. Focus groups were conducted (1) with women who kept mammography appointments; (2) with women who had scheduled, but not kept, mammogram appointments; and (3) with women who had refused to have mammograms. Approximately 250 Native American women will be randomized as participants in the two approaches to increase screening rates.

## **CANCER ETIOLOGY RESEARCH TARGETING NATIVE AMERICANS (1991-1996)**

The Extramural Programs Branch, NCI, held a workshop on and issued a RFA for research on "Cancer Risks in U.S. Ethnic/Minority Populations." The Branch supports three projects at the University of New Mexico School of Medicine that address risk factors for breast and cervical cancer and for leukemia and lymphoma among New Mexico populations including American Indians.

## **STUDIES OF HTLV IN NATIVE AMERICANS (1988-PRESENT)**

In 1988, a high prevalence of antibodies to human T-lymphotropic viruses (HTLV-I/II) was first noted in Native Americans. HTLV-I is known to cause leukemia and neurologic disease among other disorders. HTLV-II is suspected of being pathogenic but no specific disease causation has yet been proven. Through intramurally supported programs, the National Cancer Institute has expanded its studies on HTLV in several American Indian populations, documenting that HTLV-II is the predominant HTLV infecting most groups studied to date. Attention is now being focused on which diseases, if any, are caused by this virus, and which populations are most heavily infected.

## **INTRA-AGENCY AGREEMENT WITH ALASKA AREA INDIAN HEALTH SERVICE ON RISK FACTORS (1992-1995)**

The Alaska Area Indian Health Service monitors temporal trends to provide cancer incidence, mortality, and survival baseline data for Alaska Native populations. This new agreement targets specific malignancies for etiologic studies, with particular emphasis on diet and infections agents. Three malignancies are currently the focus of active research: lymphoma, with specific attention to Epstein-Barr virus; hepatocellular carcinoma, focusing on chemical carcinogenesis and hepatitis viruses; and cervical cancer, focusing on the possible interactions of human papilloma viruses and HTLV-II.

# RISK FACTORS FOR CERVICAL DYSPLASIA IN SOUTHWESTERN AMERICAN INDIAN WOMEN: A PILOT STUDY

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## ABSTRACT

Cervical cancer and cervical dysplasia occur at high rates among American Indian women in the southwestern United States. Few published data, however, have addressed risk factors for the development of cervical neoplasia among southwestern American Indian women. To investigate risk factors for cervical dysplasia in this population, we carried out a case-control pilot study focused on the effects of sexually transmitted diseases, sexual behavior, hygienic practices, cigarette use, contraceptive techniques, and diet in the development of cervical dysplasia. Although our pilot study lacked power to clearly identify risk factors for neoplasia, the data suggest that cervical papillomavirus infection (crude odds ratio 4.72, 95% confidence interval 1.62-14.11), vaginal deliveries (3.70, 0.69-20.04 for >2 vaginal deliveries vs none), and current cigarette smoking (3.08, 0.50-24.15) were associated with dysplasia. These preliminary findings indicate that risk factors for dysplasia in American Indian women differ from risks which we have identified in southwestern Hispanic and non-Hispanic white women, and suggest the need for further investigation of ethnic differences in cervical disease development.

## INTRODUCTION

The incidence of cervical cancer among minority women in the U.S. is high compared with the white majority population. For southwestern American Indian women in particular, age-adjusted incidence rates for cervical cancer are among the highest rates reported nationwide (1,2). Elevated incidence rates for cervical cancer have also been reported among American Indian women in western Washington and among American Indians/Alaska Natives in Alaska (3,4,5). Published data on incidence rates for pre-invasive dysplastic cervical lesions among American Indian women are not widely available. However, in the 1970s, Jordan and Key (6) showed high proportions of southwestern American Indian women with cervical dysplasia on routine cytologic screening tests.

Despite the excesses in cervical cancer incidence among tribal peoples, few studies have addressed the risk factors for cervical neoplasia among American Indians. In earlier investigations of pre-invasive cervical lesions, we have documented a lower prevalence of cervical human papillomavirus (HPV) infection among southwestern American Indian women compared with whites (7). We have also reported that low dietary intakes of Vitamins C, E, and folate were associated with cervical dysplasia in southwestern American Indian women (8). Other risk factor data relevant to the development of cervical neoplasia among American Indian women have not been available.

To further evaluate risks associated with pre-invasive cervical lesions among southwestern American Indian women, we carried out a pilot case-control study of risk factors for slight, moderate, and severe dysplasia among American Indian women attending a local Indian Health Service (IHS) hospital facility. These data were collected to support a grant application to comprehensively investigate risks for cervical dysplasia among American

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Indian women. Although our pilot data lack statistical power to support strong conclusions, they show the relevance and importance of further investigating risks associated with cervical neoplasia in this population.

## METHODS

### Study Subjects

Subjects were enrolled in this study through their participation in the health care delivery system of the Albuquerque Indian Health Service Hospital clinics. The clinics serve American Indian women in the Albuquerque metropolitan area and nearby reservations. Women who attend the Albuquerque IHS facilities have routine health care needs met in these facilities, including Papanicolaou (pap) smear screening tests and treatment for cervical dysplasia.

Case women were enrolled through the IHS Colposcopy Clinic before their clinical evaluation for low or high grade cervical squamous intraepithelial lesions (CIN I, II, or III; slight/moderate/severe dysplasia/carcinoma in-situ) diagnosed on routine pap smears. Colposcopy clinic visits were scheduled within a month of the diagnosis of cervical dysplasia on the routine screening pap smear. All case women were American Indian (by self-report), aged 18-40 years, and not pregnant. These women were contacted upon presentation to the colposcopy clinic and invited to participate in the interview as an additional part of their clinic visit. Subjects were asked to sign informed consent, to participate in interviews, to provide 20 ccs of blood for serologic assays and vitamin assays, and to undergo cervical cultures as described below. During the 6-month period of study entry, compliance with study entry among cases was high (96%), with only two refusals. Forty-eight (48) eligible case subjects completed interviews and examinations.

Control women were selected from the same clinics through which case women were referred for their colposcopic exams and were frequency-matched to the cases by 5-year age group. Control women were also American Indian, aged 18-40 years, and not pregnant. In addition, controls were required to have had lifetime histories of all normal pap smears. Medical records (pap smear reports) were reviewed to ensure that potential study controls had documented normal pap smears as well as met the other study enrollment criteria. Because a high proportion of women attending these clinics do not have a telephone at home and many live in rural locations that are often difficult to locate, we did not employ a random-digit dialing scheme or a neighborhood-matching scheme in selection of control women. Instead, we randomly selected control subjects from women who presented to the IHS-affiliated clinics who required a pelvic examination for any reason. Compli-

ance with study entry among control women was also high: 90% of eligible/invited controls were enrolled. Forty (40) eligible control subjects completed interviews and examinations.

Although case women with cervical dysplasia were invited into the study based on pap smears showing squamous intraepithelial lesions, the presence of dysplasia on histologic examination of cervical tissue taken on the day of study entry was necessary for subjects to remain in the analysis. Despite our focus on enrolling women with dysplastic lesions on pap smear, 8 of the 48 (16.6%) subjects enrolled as case had only atypia or non-diagnostic results on biopsy. We have elected to focus this analysis on the group of cases with dysplasia of any degree of severity as confirmed by biopsy.

For control women who were not biopsied, we required that the pap smear on the day of study entry be negative. Women who were selected as controls but who had atypia or dysplasia on the pap smear on the day of study entry (n=4) were excluded from the analysis.

### Interviews

Study procedures included interviews focused on risk factors suggested in previous research as relevant to development of cervical dysplasia. After informed consent was obtained from study subjects, a structured interview was administered. Trained interviewers asked participants about reproductive and sexual histories, sexually transmitted diseases, hygienic practices, cervical cancer screening practices, cigarette use, and diet. Demographic data were also collected. Dietary data were collected using a food frequency interview based on pilot data that described usual food items included in diets of clinic attendees (9). Food portions, as well as frequency, were estimated using food models (10). Interviews were carried out in English and lasted 60-90 minutes. The interviewers were not blinded to case or control status of the study subjects. Medical records were examined to validate responses about episodes of STDs, pap smear screening, and contraceptive use.

### Pelvic Examination and Specimen Collection

Pelvic examinations and cervical specimen collection preceded colposcopic examination of the cervix. Specimens were collected on all women in the following order: pap smears, fixed and air dried; dacron cervical swab of the endo- and ectocervix, placed in Virapap (Digene Diagnostics, Silver Spring, Maryland) transport media for later identification of HPV genome; endocervical swab for identification of chlamydia trachomatis by enzyme immunoassay (11); endocervical swab for culture of neisseria gonorrhea on Thayer Martin media (12); vaginal pool swab for culture of trichomonas vaginalis in broth (13); vaginal pool swab for culture of gardnerella vaginalis on HBT media (14);

vaginal pool swab for culture of candida on sheep red blood cell media (15); vaginal pool swab for wet-mount identification of trichomonads, yeast, and clue cells under light microscopy; and cervical-vaginal lavage using 3 cc's of normal saline (this specimen was stored for future analyses). Twenty (20) cc's of blood were collected and serum was stored at -70° C for later identification of antibodies to HSV-1 and 2 using purified glycoprotein assays (16,17). Specimens were also analyzed for levels of micronutrients as described below. Laboratory personnel were not informed as to case or control status of the subjects.

Following cervical and blood specimen collection, study subjects underwent colposcopic examination of the cervix, which had been treated with 5% acetic acid via cotton swabs. All case women underwent cervical biopsy and endocervical curettage. Treatment for any abnormal conditions was provided by physicians and staff at the IHS Hospital.

### Identification of HPV

HPV genome was identified using 2 different methods: through a commercial dot hybridization assay, Profile (Digene Diagnostics, Silver Spring, Maryland), and through the use of HPV L1 consensus primer polymerase chain reaction techniques (18,19). Profile is a new version of the more widely-available dot hybridization assay, ViraPap, and varies from the FDA approved ViraPap through inclusion of probes to detect "low risk" HPV types (6, 11, 42, 43 and 44) and "high risk" types (16, 18, 31, 33, 35, 45, 51, 52, and 56). PCR techniques were employed using the method of Manos et al. (18) (19) with the following exception: two percent of the generic-probe positive amplification product was applied to replicate nylon membranes for hybridization with probes for HPV types 6/11, 16, 18, 31, 33, 35, 39, 42, 45, 51, 52, 53, 54, 56, and 58. All specimens were processed at the University of New Mexico HPV laboratory.

### Serum micronutrient assays

Blood was obtained from study subjects for analysis of red blood cell folate (20), plasma Vitamin C (21), and Vitamin E and retinol using HPLC techniques as previously described (22). Specimens were kept on ice before transport to the laboratory for processing. All sera were stored at -70° C until assays were carried out at the UNM Clinical Nutrition Laboratory. Four (4) study subjects did not provide a specimen for micronutrient assays

### Statistical Analysis

All analyses were performed using standard packages from SAS (23). Because of the small sample size in this pilot study, we combined all cases of cervical dysplasia in one case group.

We calculated crude odds ratios as the measure of association for estimating effects of exposures and present 95% confidence limits around the point estimates of effect (24).

### RESULTS

Demographic data (Table 1) indicate that the study subjects in both case and control groups were young, and the majority had family incomes of less than \$20,000 per year. Although a larger proportion of cases listed current residence at a Pueblo or reservation compared with control women, comparable proportions of cases and

**Table 1**  
**Demographic characteristics of American Indian participants, pilot case-control study of cervical dysplasia, 1992.**

Reason for screening visit		Controls (n=36)		Cases (n=40)	
family planning	1	(2.8%)	1	(2.5%)	
annual exam	29	(80.6%)	3	(7.5%)	
other (including planned follow-up)	6	(16.6%)	36	(90.0%)	
Age at study entry (years)					
mean		29.8		26.4	
median		30		26	
Marital status					
single	11	(30.6%)	15	(37.5%)	
married	12	(33.3%)	4	(10.0%)	
divorced or separated	4	(11.1%)	2	(5.0%)	
widowed	1	(2.8%)	1	(2.5%)	
living with sex partner	8	(22.2%)	18	(45.0%)	
Current residence					
urban Albuquerque	30	(83.3%)	26	(65.0%)	
other urban	1	(2.8%)	0	--	
Pueblo/reservation	4	(11.1%)	13	(32.5%)	
other	1	(2.8%)	1	(2.5%)	
Usual residence					
urban Albuquerque	8	(22.2%)	9	(22.5%)	
other urban	3	(8.3%)	2	(5.0%)	
Pueblo/reservation	18	(50.0%)	17	(42.5%)	
other	7	(19.4%)	12	(30.0%)	
Years of education					
mean		13.9 yrs		13 yrs	
median		14 yrs		13 yrs	
range		10 -> 19 yrs		7 -> 17 yrs	
Annual family income					
< \$10,000		(27.8%)	18	(45.0%)	
\$10,000 - 19,999		(30.6%)	17	(42.5%)	
\$20,000 - 29,999		(36.1%)	3	(7.5%)	
\$30,000		(5.6%)	2	(5.0%)	



Table 2

**Risk factors for cervical dysplasia among American Indian women,  
pilot case-control study of cervical dysplasia, 1992.**

Exposure	Controls (n = 36)		Cases (n = 40)		Crude OR (95% CI)	
Age at first intercourse						
20+	7	(19.4%)	6	(15.0%)	1.00	
16-19	21	(58.3%)	21	(52.5%)	1.16	(0.34, 4.06)
<16	8	(22.2%)	13	(32.5%)	1.90	(0.47, 7.70)
Lifetime no. of sex partners						
1	5	(13.9%)	7	(17.5%)	1.00	
2 - 5	19	(52.7%)	13	(32.5%)	0.49	(0.12-1.88)
6 - 10	12	(33.3%)	20	(50.0%)	1.19	(0.30, 4.67)
History of smoking						
Never	22	(61.1%)	25	(62.5%)	1.00	
Past	12	(33.3%)	8	(20.0%)	0.59	(0.20, 1.70)
Current	2	(5.6%)	7	(17.5%)	3.08	(0.50, 24.15)
History of O.C. use						
Never	7	(19.4%)	5	(12.8%)	1.00	
Past	14	(38.9%)	14	(35.9%)	1.40	(0.36, 5.49)
Current	15	(41.6%)	20	(51.3%)	1.87	(0.41, 8.64)
Vaginal Deliveries						
0	14	(38.9%)	17	(42.5%)	1.00	
1	12	(33.3%)	10	(25.0%)	0.68	(0.23, 2.07)
2	8	(22.2%)	4	(10.0%)	0.41	(0.10, 0.65)
> 2	2	(5.5%)	9	(22.5%)	3.70	(0.69, 20.04)

controls listed their usual place of residence on Pueblo/ reservations lands. Years of formal education were comparable between groups.

We collected information on sexual behavioral variables that have been associated with cervical dysplasia in other populations (Table 2). Early age at first intercourse was not associated with dysplasia in this study. A history of having had more than 10 lifetime sex partners was associated with dysplasia, although this finding was not statistically significant. Other risks associated with dysplasia are shown in Tables 2 and 3. For most risk factors examined, power to show statistically significant differences was limited by sample size. Despite the small sample size, however, crude risks associated with HPV as identified by Profile and by PCR were statistically significant (Table 3). The prevalence of HPV by type and by case-control status is shown in Table 4.

Statistically significant differences in plasma micronutrients and in red blood cell folate levels were not apparent. For each of these assays, standard deviations were large and mean differences between case and control groups were small (Table 5).

## DISCUSSION

Risk factors for cervical neoplasia vary among populations worldwide, although the most consistently-rec-

ognized risks for cervical neoplasia include cervical HPV infection, early age at first sexual intercourse, high number of sex partners, cigarette smoking, parity, low economic status, few years of education, male partner promiscuity, oral contraceptive use, and lack of frequent cytologic abnormalities (25,26). The data are less consistent among various studies in reference to the roles of: genital herpesvirus infections; other STDs; low dietary intakes of vitamins A, C, E, and folate; low serum micronutrient values for vitamins A, C, E, and folate; douching; and frequency of intercourse (25,26). Racial and ethnic factors also appear to influence cervical neoplasia risk: worldwide, the highest rates of invasive cervical cancer have been reported from Latin America (26-28), although American Indian women have shown great excesses in both cervical cancer incidence (3,5,7) and mortality (7,29,30).

Our pilot study was designed to collect data on numerous factors as potentially related to cervical dysplasia in southwestern American Indian women. Although this project was not undertaken to definitively characterize risks associated with cervical dysplasia in this population, the data suggest that several factors may be associated with dysplasia in this group. Consistent with other reports on cervical neoplasia risk (31), our findings suggest associations with cervical HPV infection and cervical dysplasia. Although cervical HPV

Table 3

**Laboratory data for pilot case-control study of cervical dysplasia among  
American Indian women, 1992.**

Exposure	Controls (n = 36)		Cases (n = 40)		Crude OR (95% CI)	
HPV by Profile						
neg	32	(88.9%)	26	(65%)	1.00	
pos	4	(11.1%)	14	(35%)	4.31	(1.13-17.81)
HPV by PCR						
neg	25	(69.4%)	13	(32.5%)	1.00	
pos	11	(30.6%)	27	(67.5%)	4.72	(1.62-14.11)
HSV-1 Antibodies						
neg	11	(30.6%)	6	(15.8%)	1.00	
pos	25	(69.4%)	32	(84.2%)	2.24	(0.76-7.22)
HSV-2 Antibodies						
neg	25	(69.4%)	26	(68.4%)	1.00	
pos	11	(30.6%)	12	(31.6%)	1.04	(0.39-2.83)
GC culture						
neg	36	(100%)	40	(100%)	--	
pos	0	--	0	--	--	
gardnerella culture						
neg	14	(38.9%)	17	(42.5%)	1.00	
pos	14	(38.9%)	14	(35%)	1.21	(0.43, 3.41)
specimen inadequate	8	(22.2%)	9	(22.5%)		
trichomonas culture						
neg	36	(100%)	38	(95%)	--	
pos	0	--	2	(5%)	--	
chlamydia*						
neg	34	(94.4%)	38	(95%)	1.00	
pos	2	(5.6%)	1	(2.5%)	0.45	(0.04-4.95)
specimen inadequate			1	(2.5%)		
trichomonas wet prep						
neg	34	(97.1%)	36	(94.7%)	1.00	
pos	1	(2.9%)	2	(5.3%)	1.89	(0.16-21.79)
yeast wet prep						
neg	33	(94.3%)	37	(97.4%)	1.00	
pos	2	(5.7%)	1	(2.6%)	0.45	(0.04-5.15)

\*enzyme immuno-assay

infection has been consistently reported to be a risk factor for cervical neoplasia in other populations, the risk estimates vary substantially depending on the HPV detection method employed (32,26,19), and by disease outcome under investigation (dysplasia vs carcinoma-in-situ vs invasive cancer). We used a newly-developed version of a dot hybridization assay (Profile); thus, comparison data from other studies of dysplasia are not yet available, although the test kit we used is similar to the earlier-developed ViraPap HPV detection test. We have recently completed a case-control study of risks for cervical dysplasia among New Mexico Hispanic and non-Hispanic white women using ViraPap as one HPV detection method. We found that the risk of high-grade cervical dysplasia among HPV-infected women was 12.4 (95% confidence interval, 8.0-19.4) compared to non-infected women. PCR-based HPV detection methods showed even higher risk estimates—19.4 (10.1-37.9) for infected vs non-infected subjects. We also

found major ethnic differences in disease risk associated with HPV 16/18 infection as detected by ViraType tests. The adjusted odds ratio for non-Hispanic white women was 15.7 (5.6-44.5), but for Hispanics was 168.1 (22.4-1260.7) for HPV 16/18 in association with dysplasia (unpublished data). We plan to further examine HPV-associated risks in American Indian women in New Mexico to determine if ethnic differences in the magnitude of risk are also apparent in that ethnic/racial group.

Although cigarette smoking is not widely prevalent among American Indians in the Southwest (33), and rates of cigarette-related cancers are very low among local Indian peoples (34,1), our data suggest that cigarette smoking at the time of diagnosis of dysplasia is a risk factor for disease. In contrast, remote cigarette use was not strongly associated with dysplasia. These observations are consistent with results from many case-control studies of cervical neoplasia worldwide and support theories of late-stage effects of cigarette-related



Table 4

**Prevalence of HPV types by case/control status, pilot study of cervical dysplasia among American Indian women, 1992.**

	Controls (n = 36)	Cases (n = 40)
	No (%) Pos	No (%) Pos
PCR Assays		
Type 6/11	1 (2.8)	4 (10)
Type 16	1 (2.8)	8 (20)
Type 18	0	1 (2.5)
Type 31	0	2 (5.0)
Type 33	0	0
Type 35	1 (2.8)	0
Type 39	1 (2.8)	2 (5.0)
Type 42	0	0
Type 45	0	0
Type 51	0	1
Type 52	0	0
Type 56	0	6 (15)
Type 58	1	2 (5.0)
Generic probe	11 (30.6)	27 (67.5)
Profile		
Low risk	1 (2.8)	5 (12.5)
High risk	3 (8.3)	11 (27.5)
High or low	4 (11.1)	14 (35.0)

mutagens in development of neoplastic lesions. In our study of risk factors for high-grade cervical dysplasia in local Hispanic and non-Hispanic white women, we found comparable risk estimates for both remote and current cigarette use as we have shown for American Indian women in this study (35).

We expected to observe differences in serum micronutrient values between case and control groups; the data that we present (Table 5) did not support our hypotheses that low serum micronutrient levels would be associated with cervical dysplasia. Using 24-hour food recall data in a case-control study of micronutrients and cervical dysplasia among local American Indian women, we documented low folacin (RR=3.0 for low vs high intake), low vitamin C (RR=3.0), and low vitamin E (RR=1.7) intakes as risks for slight moderate, or severe dysplasia (08). Other data have also documented micronutrient deficiencies in high proportions of the state's American Indians and have shown that dietary patterns and nutrient intakes of American Indians in the Southwest vary significantly from non-Indian peoples. (36,37,38) The lack of differences between mean micronutrient values of case and control subjects in this study may reflect study subjects' easy access to fruits and vegetables during the summer months when we collected these data. In our proposed comprehensive study, we will collect data from study subjects during all seasons for a three-year period and will be able to

Table 5

**Micronutrient data for case and control subjects, pilot study of cervical dysplasia among American Indian women, 1992.**

Exposure	Controls (n = 32)	Cases (n = 39)
RBC folate (ng/ml)		
mean	330.3	320.3
median	300.5	297.5
range	162.0-660.0	162.0-700.0
Vitamin C (mg%)		
mean	0.97	0.85
median	0.97	0.83
range	0.32-2.4	0.31-1.48
Retinol (μ/ml)		
mean	0.53	0.54
median	0.54	0.52
range	0.35-0.78	0.27-0.83
Vitamin E (μ/ml)		
mean	8.22	7.68
median	7.69	7.68
range	9.93-17.56	9.97-10.95

identify season-specific differences in micronutrient serum levels and in micronutrient intakes between case and control groups.

Our pilot data support previously-reported associations of oral contraceptive use and of parity as risks for cervical neoplasia. The exact role of oral contraceptive use in the development of cervical neoplasia is not clear (39), as epithelial cell cancers are not generally thought to be strongly influenced by hormonal factors. The etiologic role of multiparity in cervical disease development is also not widely agreed upon by scholars in the field. Hormonal influences on cervical epithelium, pregnancy-induced immunosuppression, and trauma related to vaginal deliveries have all been suggested as factors related to development of neoplasia of the cervix (39). In our case-control study of cervical dysplasia among Hispanic and non-Hispanic white women, we did not observe an increase in risk associated with oral contraceptive use, but did find similar associations with multiparity as we have observed in this pilot project (40).

Our pilot study does not have adequate power to examine tribal differences in risk for dysplasia. Because diet, sexual behavior, prevalence of infections, and reproductive factors may vary substantially among tribes, such differences in disease risk may be important, especially from the standpoint of prevention programs. Although high rates of invasive cervical cancer have been

reported among different groups of American Indians/Alaska Natives in different geographic areas, we lack tribe-specific information on risk factors for invasive or pre-invasive cervical disease. Multicentered, collaborative investigations of cervical neoplasia are necessary to address the issue of tribal variation in disease risks. Such investigations should consider genetic differences in development of cervical lesions, such as variation in HLA haplotypes, which may be relevant to cervical neoplasia (41).

## LIMITATIONS

In addition to the small sample size and lack of statistical power, our investigation has other potential limitations which must be recognized. The interviewers were not blinded to case or control status of the study subjects; however, interviewer bias was minimized by using structured interviews with detailed question-by-question instructions. Although the selection of control women was not based on random sampling of all eligible clinic attendees, our approach in sampling eligible women who presented to the IHS clinics requiring a pelvic examination for any reason should not have introduced systematic error into the study design or analysis of the results. Because controls were selected from the same clinics from which cases had their screening pap smears, referral bias should not be a factor which biases our results. Recall bias and information bias are always potential problems in case-control studies involving sexual histories. To reduce possible information bias for reported sexual behavior and sexually transmitted diseases, the study subjects were reminded at several points in the interview about the confidential nature of their responses and the importance of accurate responses. Although the laboratory tests we used for identifying cervical infections were "state-of-the-art," our method of testing for HPV infection at a single point in time may not accurately reflect the true status of cervical HPV infection. We and other research groups have shown that sequential testing for cervical HPV genome in the same women substantially increases measures of prevalence of infection, even in the absence of new sex partners (42). We did not collect data on sexual behavior of male sex partners of study subjects. Sexual behavior of male partners may be relevant to the development of cervical disease among case women (43,44); we cannot evaluate the effects of such variables in this study. Last, we combined women with all degrees of dysplasia as our case group. We recognize that not all pathologists agree that cervical dysplasia represents a continuum from slight dysplasia to invasive carcinoma, and that our strategy of combining women with pre-invasive dysplasias in one category may be inappropriate. While we agree that risk factors for each degree of dysplasia

should be examined separately, our funding limitations and resultant small sample size would not permit further stratification of our case group by degree of histologic abnormality. We plan to further expand our case and control series to allow adequate analysis of risks for various degrees of dysplastic lesions among American Indian women.

Despite limitations of our data, our findings suggest that dysplasia among American Indian women in the southwest may be associated with cervical HPV infection, cigarette use, use of oral contraceptives, parity, and a high number of sex partners. Adjusted analyses, which consider relevant confounders and which are carried out in a larger sample, are needed to more definitively characterize these risks as potentially related to cervical dysplasia. This information should eventually prove useful in cervical dysplasia/cervical cancer prevention programs among American Indian women in the Southwest.

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Although this research was carried out at an Indian Health Service facility, the opinions expressed in this manuscript do not necessarily reflect those of the Indian Health Service.

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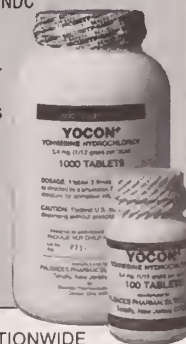
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# CANCER RISK FACTORS IN THREE SIOUX TRIBES

## Use of the Indian-Specific Health Risk Appraisal for Data Collection and Analysis

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### ABSTRACT

Cancer mortality rates for Aberdeen Area Indians exceed U.S. rates with lung cancer being the leading cause of cancer death. The Sioux Cancer Study, an ancillary study of the Strong Heart Study, investigated cancer and cancer risk factors among tribal members aged 45-74 in three Sioux tribes in North and South Dakota. An Indian-specific health risk appraisal was used to collect data and provide specific recommendations to participants. The high rates of smoking (56% for men and 48% for women) explain the high lung cancer mortality rates. Intensive smoking cessation and prevention programs will likely have the greatest impact in reducing preventable cancer deaths. More accessible cervical and breast cancer screening provided by female health care providers is needed to reduce preventable cancer deaths among Sioux women. Pap smear screening is an especially high priority since cervical cancer mortality is 4.4 times higher than U.S. rates, all races. Programs targeted to reduce obesity and excessive alcohol use will also likely reduce preventable cancer deaths associated with high rates of obesity, diabetes and binge drinking. Community-based cancer prevention and control programs tailored to the cancer risk factor profile of the community are the best strategy to reduce preventable cancer deaths in Indian communities.

### INTRODUCTION

Cancer mortality rates are higher among Aberdeen Area Indians than among Indians from the Southwest and the U.S. general population (Table 1) (1,2). Most Sioux Indians receive medical care from the Aberdeen Area Indian Health Service (AAIHS), which is responsible for providing clinical and preventive services for American Indians residing in North Dakota, South Dakota, Iowa, and Nebraska. In order to better understand the reasons for high rates of cancer and to reduce those rates, the Aberdeen Area Epidemiology Program received funding from the National Cancer Institute to establish and evaluate a cancer screening program conducted in the Oglala, the Cheyenne River, and the Devils Lake Sioux Tribes.

The Sioux Cancer Study (SCS), as this screening program is called, is a companion study of the Strong Heart Study, a study of cardiovascular disease in the same three tribes. In addition to evaluating the participants in the Strong Heart Study for factors that cause heart disease and stroke, the participants were also evaluated for factors that cause cancer and were also offered routine cancer screening.

### METHODS

The eligible population for the SCS examination includes resident members aged 45 to 74 years between July 1, 1989, and January 31, 1992, of the following tribes: The Oglala and Cheyenne River Sioux in South Dakota and the Devils Lake Sioux in the Fort Totten area of North Dakota. The study population was enumerated by review of the tribal rolls by long time residents. People who no longer lived in the study area, who were deceased, or who were institutionalized were identified removed from the list of eligible participants. Fifty-five percent of eligible tribal members participated. Preva-

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(1) Medical Epidemiologist

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(4) Coordinator

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Table 1

**NATIVE AMERICAN & OVERALL US MORTALITY\***  
1984-1988

CANCER	NATIVE AMERICAN MORTALITY				MORTALITY U.S. ALL RACES	RELATIVE RISK
	12 IHS AREAS	9 IHS AREAS''	ABERDEEN AREA'''			ABERDEEN AREA VS. US
OVERALL	131.9	148.5	198.3	(373)	171.3	1.16
MALE	133.9	142.6	192.9	(182)	218.7	0.88
FEMALE	130.1	154.1	203.5	(191)	139.5	1.46
ORAL-PHARYNGEAL	2.3	2.5	2.3	(5)	3.1	0.74
MALE	3.2	3.2	4.7	(5)	4.9	0.96
FEMALE	1.4	1.7	--	(0)	1.7	--
ESOPHAGEAL	2.0	2.2	1.8	(3)	3.4	0.53
MALE	3.0	3.2	1.1	(1)	5.8	0.19
FEMALE	1.2	1.2	2.4	(2)	1.5	1.60
STOMACH	7.7	9.9	9.5	(19)	5.0	1.90
MALE	9.1	11.0	12.1	(12)	7.3	1.66
FEMALE	6.3	8.9	6.9	(7)	3.3	2.09
COLO-RECTAL	12.0	11.2	13.3	(24)	20.1	0.66
MALE	11.6	10.6	16.3	(15)	24.5	0.66
FEMALE	12.4	11.8	10.4	(9)	17.1	0.61
LIVER	3.7	4.2	3.5	(6)	2.2	1.60
MALE	4.1	4.9	3.3	(3)	3.3	1.0
FEMALE	3.3	3.5	3.7	(3)	1.4	2.64
GALLBLADDER	3.7	5.1	5.2	(9)	0.8	6.50
MALE	1.7	2.3	3.4	(3)	0.5	6.80
FEMALE	5.6	7.6	6.8	(6)	1.0	6.80
PANCREATIC	7.4	8.3	12.5	(23)	8.4	1.49
MALE	7.8	8.7	13.3	(12)	10.1	1.32
FEMALE	6.9	7.8	11.8	(11)	7.2	1.64
LUNG	30.5	32.7	56.5	(104)	47.1	1.20
MALE	40.1	38.5	68.7	(63)	74.2	0.93
FEMALE	21.4	27.2	45.0	(41)	27.3	1.65
RENAL	4.4	5.1	5.2	(11)	3.3	1.58
MALE	5.6	6.6	10.2	(10)	4.8	2.13
FEMALE	3.2	3.6	0.4	(1)	2.2	0.18
BREAST	14.6	16.1	24.0	(23)	27.3	0.88
CERVICAL	7.6	10.0	13.6	(16)	3.1	4.39
UTERINE	1.7	1.5	6.5	(6)	1.9	3.42
OVARIAN	6.7	7.4	7.2	(7)	7.7	0.94
TESTES	0.4	0.7	0.5	(1)	0.3	1.67
PENIS	0.1	0.2	2.2	(2)	0.2	11.0
PROSTATE	14.4	15.8	25.4	(23)	24.0	1.06
HODGKIN'S	0.4	0.7	--	(0)	0.7	--
MALE	0.3	0.2	--	(0)	0.8	--
FEMALE	0.6	1.2	--	(0)	0.5	--
NON-HODGKIN'S	3.5	3.2	5.7	(10)	5.8	0.98
MALE	3.4	3.3	1.1	(1)	7.0	0.16
FEMALE	3.6	3.0	10.0	(9)	4.8	2.08
MULTIPLE MYELOMA	2.8	3.2	6.7	(11)	2.8	2.40
MALE	2.2	2.5	2.3	(2)	3.5	0.66
FEMALE	3.4	3.8	10.8	(9)	2.4	4.5
LEUKEMIA	4.2	4.4	7.0	(14)	6.4	1.09
MALE	4.3	4.0	7.3	(7)	8.4	0.87
FEMALE	4.1	4.8	6.6	(7)	5.0	1.32
ILL-DEFINED	13.6	16.9	16.2	(29)	12.6	1.29
MALE	12.7	15.5	12.4	(12)	15.8	0.78
FEMALE	14.5	18.3	19.7	(17)	10.3	1.91

\*\*\* Numbers in parentheses represent total deaths 1984-1988.

\*\* 3 IHS Areas (California, Oklahoma, and Portland) have a problem with underreporting of Indian race on death certificates and these rates include the other 9 IHS areas where vital records are more accurate

\* Taken from Cancer Mortality among Native Americans in the United States. An IHS report, 1992.  
Average annual rates per 100,000 population age-adjusted to 1970 US population.

lence of cancer risk factors are calculated using as the denominator all tribal members who attended the clinical exam. The study protocol was approved by the Indian Health Service Institutional Review Board (IRB) and by the participating tribes.

The clinical examinations, consisting of a personal interview, physical examination, and laboratory tests, were conducted as an ancillary study to the Strong Heart Study, which is a study of cardiovascular disease in American Indians. The design and methods of the Strong Heart Study have been described elsewhere (3). After informed consent was obtained, interviews were conducted. Most of the cancer screening exams were done by female nurse practitioners. These exams consisted of pelvic exams, pap smears, breast exams, and rectal and testicular exams. Mammograms were provided by a contract mobile unit that offered screening mammograms to SCS participants in some of the participating communities at a different time. Some of the data were collected using the Indian-specific Health Risk Appraisals (HRA), and all participants received the recommendations on ways to improve their health based on their individual HRA computer analysis (4,5). The participants completed the HRA themselves. Project staff provided assistance when needed and insured that data entry was complete.

Anthropometric measurements were made with shoes removed, and with participants wearing lightweight clothing. Questions administered during the interview assessed demographic information, family health history, lifestyle, and medical history. Since almost all participants were fluent in English, the interview questions were read in English. Native language was used when needed for participant comprehension.

Standardized protocols and data collection forms were used at each field site. Obesity was defined as 120% of ideal body weight using the 1959 Metropolitan Life Insurance Company height/weight tables, which are incorporated into the HRA software (6).

## RESULTS

### A. Sociodemographic Factors (Table 2)

There were 1538 participants in the Sioux Cancer Study. Overall 55% of the enrolled tribal members were residing on or near the reservation. The average age of participants was 56.0 years with male participants 0.3 years younger than females. The mean ages were the same for the three tribes.

Less than half of the participants had a high school education. About one third of the participants were employed full time or part time.

### B. Tobacco and Alcohol Use and Seat Belt Non-use (Table 3)

In all three tribes, a higher proportion of men smoked than women, and male smokers smoked more cigarettes per day than female smokers. Less than 2 percent of the participants reported smoking cigars or pipes. Smokeless tobacco use was uncommon with less than 2 percent of the participants reporting daily use. Only 5 females (0.6 percent) reported using smokeless tobacco.

Less than half the participants reported using alcohol in the last month. One fourth of those surveyed admitted to having one or more binges in the last month as defined by five or more drinks on an occasion. Binge drinking was reported more often among men than women. Almost 60 percent of participants rarely use seatbelts and only 12 percent almost always use them.

### C. Obesity, Family History of Diabetes, Diabetes and Sedentary lifestyle (Table 4)

About 2/3 of the men and 3/4 of the women exceed 120 percent of ideal body weight. Half of the participants reported a family history of diabetes (mother, father, brothers and/or sisters). Overall 28 percent of the participants reported they had been told they have diabetes or sugar diabetes, with women reporting diabetes more often than men (32.2 percent vs 21.4 percent). Most participants reported participating in leisure time exercise less than three times per week.

### D. Cancer Screening (Table 5)

Although most men and women reported having had a rectal exam in the last two years, 16 percent reported that they had never had a rectal exam.

Average age of menarche was 13.5 years, and 44 percent of women had their first child as a teenager.

Although most women had at least one mammogram, 38 percent reported never having had one. A higher proportion of Cheyenne River Sioux women reported having had a mammogram. Eleven percent of the women reported a family history of breast cancer (mothers or sisters only) with 15 percent of Cheyenne River Sioux women reporting such a history. Over 40 percent of the women perform monthly self-breast exams, and over half reported having had a clinical breast exam in the last two years.

A third of the women had not had a pap smear in the last three years, and 4 percent of women said they never had a pap smear. Pap smear screening rates are similar in all three tribes.



Table 2		Sociodemographic Factors			
		Oglala Sioux	Cheyenne River Sioux	Devils Lake Sioux	TOTAL
<b>Total Number of Participants</b>		884	430	218	1532
Percent of Males		43.9	39.7	43.1	42.6
Percent of Females		56.1	60.3	56.9	57.4
Percent of High School Graduates		46.7	48.6	37.0	45.9
Percent of Employment Status	Full Time	32.6	28.1	38.4	32.2
	Part time	5.9	6.1	6.5	6.0
	Total	38.5	34.2	44.9	38.2

Table 3		PREVALENCE OF TOBACCO AND ALCOHOL USE			
		Oglala Sioux	Cheyenne River Sioux	Devils Lake Sioux	TOTAL
<b>Tobacco Use</b>					
Current Cigarette Users (percent)	Males	55.1	55.0	59.6	55.7
	Females	47.2	46.3	53.2	47.8
	Total	50.7	49.8	56.0	51.2
Average Number of Cigarettes Smoked Per/Day	Males	13.7	15.5	15.7	14.5
	Females	11.3	11.2	12.8	11.5
	Total	12.4	13.1	14.2	12.9
Percent of Current Smokeless Tobacco Users		1.6	1.6	2.3	1.7
<b>Alcohol Use</b>					
Current Users (percent)					
≥ 1 Drink/Week		28.1	20.6	22.9	25.3
≥ 1 Day/Month		36.4	28.3	27.5	32.9
≥ 5 Drinks/Occasion in past Month	Males	36.3	31.5	33.3	36.4
	Females	17.9	13.7	12.2	15.9
	Total	27.3	20.8	21.3	24.6
Current Users (Means)					
≥ 1 Drink/Week		9.6	11.5	9.9	10.1
≥ 1 Day/Month		6.3	5.8	7.1	6.3
Ride or Drive After Drinking In Last Month (percent)		9.9	7.0	8.3	8.8
<b>Safety Belt Use (percent)</b>					
Rarely		53.7	63.9	69.4	58.8
Almost Always		13.7	9.6	13.4	12.5

## DISCUSSION

The Sioux Cancer Study demonstrated that the HRA can be a useful data collection tool for research studies as well as for facilitating health education in study participants. The HRA group summary analyzed HRA data in a format that is easy to use for community studies. This is especially important in research conducted in

Indian communities since frequently the research participants and the communities do not realize immediate benefits (7). Almost all Sioux Cancer study participants were given the results of their HRA appraisal and provided a personalized interpretation before leaving the exam site. Although many have informally reported making positive behavior changes based on the counseling they received, the effectiveness of the HRA in

Table 4

**Prevalence of: Obesity, Family History of Diabetes;  
Diabetes and Sedentary Lifestyle**

		Oglala Sioux	Cheyenne River Sioux	Devils Lake Sioux	TOTAL
<b>Body Wt. in Pounds (Means)</b>	Males	190.1	194.2	199.2	192.4
	Females	170.7	174.6	171.0	171.9
	<b>Total</b>	<b>179.2</b>	<b>182.4</b>	<b>183.1</b>	<b>180.7</b>
<b>Obesity &gt; 120% of Ideal Body Wt. (percent)</b>	Males	68.7	74.8	65.6	69.9
	Females	77.4	78.8	75.0	77.5
	<b>Total</b>	<b>73.6</b>	<b>77.3</b>	<b>71.0</b>	<b>74.2</b>
<b>Family Members with Diabetes (percent)</b>	Total	48.7	57.6	57.6	52.4
<b>History of Diabetes (percent)</b>	Males	19.5	20.5	30.8	21.4
	Females	31.5	27.8	44.4	32.2
	<b>Total</b>	<b>26.2</b>	<b>24.9</b>	<b>38.5</b>	<b>27.6</b>
<b>Exercise &lt;3 Times/Week (percent)</b>	Total	58.7	47.9	67.0	56.8

Table 5

**Cancer Screening**

		Oglala Sioux	Cheyenne River Sioux	Devils Lake Sioux	TOTAL
Years Since Last Rectal Exam (percent)	< 1 Yr.	28.0	31.7	25.6	28.7
	1 Yr.	15.0	11.5	12.1	13.6
	2 Yr.	10.3	10.8	10.7	10.5
	≥ 3 Yr.	30.2	30.3	34.4	30.8
	Never	16.5	15.7	17.2	16.4
<b>WOMEN ONLY</b>					
Average Age of 1st Menstrual Period		13.7	13.3	13.3	13.5
Had 1st Child < Age 20 (percent)			42.3	45.7	49.2
44.3					
Years Since Last Mammogram (percent)	< 1 Yr.	19.9	22.6	30.3	22.1
	1 Yr.	12.7	13.6	10.1	12.6
	2 Yr.	8.4	7.4	2.5	7.3
	≥ 3 Yr.	16.9	27.2	15.1	19.7
	Never	42.1	29.2	42.0	38.3
Family History of Breast Cancer (percent)	0	88.5	82.5	86.0	86.4
	1	7.8	14.8	10.7	10.3
	≥ 2	0.4	0.4	0.8	0.5
	Don't Know	3.2	2.3	2.5	2.9
Self Breast Exam (percent)	Monthly	40.0	42.4	47.2	41.7
	Every Few Months	19.6	22.2	14.6	19.7
	Rarely or Never	40.4	35.4	38.2	38.6
Years Since Last Clinic Breast Exam (percent)	<1 Yr.	37.9	37.7	39.0	38.0
	1 Yr.	17.7	18.7	13.8	17.5
	2 Yr.	11.1	8.6	8.1	9.9
	≥ 3 Yr.	21.4	28.4	22.0	23.5
	Never	11.9	6.6	17.1	11.1
Years Since Last Pap Smear (percent)	<1 Yr.	31.7	35.7	31.2	32.8
	1 Yr.	20.0	17.4	18.0	19.0
	2 Yr.	13.5	11.2	12.3	12.7
	≥ 3 Yr.	30.1	32.6	34.4	31.4
	Never	4.6	3.1	4.1	4.1



motivating behavior change in Indian communities has not been evaluated. Preliminary investigations as well as feedback from HRA users suggest that a number of revisions are needed to increase the effectiveness of the HRA as a health promotion/disease prevention tool (8). The AAIHS Epidemiology Program has submitted a proposal to evaluate and update the Indian-specific HRA.

The high unemployment and low level of education of the participants probably contributes to the high rates of cancer mortality (9). Cancer prevention and control programs in this population must therefore consider the poverty and educational levels of the patients in order to be successful.

Rates of cigarette smoking are high in all three tribes. The high smoking rates likely contribute to their high rates of lung cancer as well as the high rates of cardiovascular disease and chronic obstructive pulmonary disease (10). More intense smoking cessation and prevention programs are being implemented in Indian communities (11), and many tribal nations are developing smoking policies that reduce environmental tobacco smoke exposure in public buildings in accordance with a recent EPA advisory (12,13).

High rates of smokeless tobacco use have been reported in Indian children and adolescents (14), but few SCS participants used smokeless tobacco, pipes, or cigars.

While moderate alcohol use has generally not been found to be a risk factor for cancer, alcohol abuse and intoxication may be a risk factor for some types of cancer (liver, esophagus, and breast) (15,16). Alcohol-related morbidity and mortality are high in most Indian populations, causing high rates of premature loss of life as measured by the years of potential life lost (YPLL) (17). Non-use of seat belts is also a factor in these higher rates of premature death. Although the prevalence of alcohol use in SCS participants is less than the 1990 national rates (63 percent for adult males 45 years and over and 40.8 percent for adult females 45 years of age and older) (18), the pattern of heavy alcohol consumption and binge drinking that occurs commonly among AI/AN is associated with many adverse health events, including sudden death (19). The median prevalence rate of binge drinking in the last month among persons aged 50-64 in 36 states participating in CDC's behavioral risk factor survey in 1988 was 8.1 percent (20), a rate that is also lower than the rates we found in SCS participants in all three tribes.

Increased support for substance abuse treatment and prevention is available for Indian communities and if successful will have a positive impact on health-related problems afflicting Indians including cancer.

In the last 100 years drastic changes in lifestyle have occurred in Indian communities including a less active lifestyle and consumption of a higher fat, lower fiber diet. Obesity has become increasingly prevalent among native groups and is a major determinant of

diabetes in the Pima Indians (21,22), especially those with a family history of diabetes. Although the association of obesity and cancer in Indian populations has not been investigated, obesity has been associated with shorter survival from breast cancer in other populations, and its role in the poor survival of Indian women with breast cancer should be investigated (16). Sedentary lifestyles and obesity may also increase the risk of colon cancer.

Although the epidemic of diabetes has been associated with high rates of complications, including end stage renal disease, retinopathy, and amputations, its association with cancer is not well defined. Obesity has been reported to increase the risk of uterine cancer in other populations (24). Further study of the association between obesity, diabetes, and cancer in Indian populations is needed.

Funding has been made available through the National Heart, Lung and Blood Institute to determine the best ways to prevent obesity in Indian children (23). Community-based programs that promote healthier diets and physical activities that are in keeping with Indian traditional values and cultures have the greatest likelihood of success in preventing obesity, thus reducing the risk of developing diabetes, cancer, and other chronic diseases (24,25).

Because the rates of cervical cancer mortality are so high in this population and because 1/3 of women aged 45-74 in these three tribes have not been screened in the last three years, more intensive screening programs are needed to reduce the barriers to participation (26). Female providers and availability of screening at the nearest health facilities are key factors in successful screening programs.

Mobile mammography units that could also perform cervical cancer screening and cancer screening for men (one-stop shopping concept) may be the best strategy for reducing barriers to cancer screening in rural Indian communities. Such units could visit each community annually to provide recommended screening in conjunction with a community education program conducted by community health representatives and health educators.

Knowledge, attitudes, and beliefs about cervical and breast cancer of SCS participants and the costs and results of the cancer screening conducted as part of the study are reported elsewhere (27,28).

Patterns of cancer vary greatly among Indian tribes, probably due to differences in cancer risk factor prevalences and possibly due to differences in genetic susceptibility to various types of cancer (29).

## CONCLUSION

The Indian-specific HRA was a valuable tool for the SCS to collect data, to provide group summaries, and to

educate participants on health promotion and disease prevention. The high mortality cancer rates among the Sioux are likely related to high rates of smoking, alcohol abuse, sedentary lifestyle, obesity and diabetes as well as inadequate screening and lack of accessibility to state of the art cancer treatment. More accessible cancer screening with female providers is needed to reduce the rates of cervical and breast cancer. Community-based cancer prevention and control programs offer the best hope of success in reducing preventable cancer mortality. Such programs should be developed as comprehensive programs that will have the greatest impact on all chronic diseases afflicting Indians.

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# PATHWAYS TO HEALTH

## A HEALTH PROMOTION AND CANCER PREVENTION

### PROJECT FOR AMERICAN INDIAN YOUTH

Leslie Cunningham-Sabo, MS, RD, CHES  
Sally M. Davis, PhD, CHES

#### INTRODUCTION

While cancer incidence rates among American Indians are generally lower than those of the general population, cancer remains the third leading cause of death among American Indians of all ages (1). Differences in cancer survival rates are largely attributable to detrimental socioeconomic or environmental factors rather than race (2), as the percent of American Indians living below the poverty level is more than two times that for the entire U.S. (2).

The Centers for Disease Control and Prevention have reported that approximately half of all factors which influence an individual's chances of surviving to age 65 are related to lifestyle behaviors). About 30% of all cancer deaths are related to tobacco use (3), and about 50% of cancer incidence is related to diet (4). Numerous research articles link specific foods or nutrients to certain types of cancer (5). Lifestyle behavioral goals of both the National Cancer Institute and the American Cancer Society include avoidance of all tobacco products, decreased consumption of dietary fat (to less than 30% of daily calories), and increased consumption of dietary fiber (to 20-30 grams per day) (6,7). While results from studies linking obesity and cancer are not entirely consistent, maintenance of desirable weight is recommended and may potentially decrease the risk for certain cancers (8).

Recent studies of American Indian children in Arizona and New Mexico have documented a significant rate of obesity among these tribal groups (9-11). Like children elsewhere in the U.S. (12), Navajo and Pueblo children's dietary composition reflects the nation's as a whole, with approximately 35% of daily calories from fat (11). In addition, rates of both cigarette and smokeless tobacco use among Native American adolescents in New Mexico are higher than rates for other New Mexican youth (13).

Given children's preference for high-fat foods (14) and continued use of tobacco products, we recognize that their social environment needs to be modified in order to expose them to more healthful alternatives. Potentially schools can do more than any other single agency in society to help young people develop healthful lifestyle behaviors (15). These behaviors can foster improved school performance, reduced dropout rates,

and better health as an adult. However, one must keep in mind that cultural beliefs and practices exert strong influences on decisions made in daily living, including those choices related to personal health (16). For Indian youth, desired health behaviors must be modeled and demonstrated within a cultural context to be useful in bringing about favorable lifestyle changes (17).

This manuscript describes the development, implementation, and initial evaluation of a school-based health promotion and cancer prevention project for Southwestern Indian youth and their families called Pathways to Health. Preliminary dietary results from a pilot test of this project are described here. Additional results are described elsewhere (18).

#### PATHWAYS TO HEALTH PROJECT

##### Population

There are nineteen Pueblo communities in New Mexico, mainly near the Rio Grande and its' tributaries in the north-central portion of the state. Zuni Pueblo is located centrally near the Arizona border. Navajo lands stretch from the northwest section of the state into Arizona and Utah.

Pathways to Health was initiated in nine Navajo and Pueblo schools in rural, northwest New Mexico at the request of school and community members. Schools were selected as the intervention site for two reasons: they can offer appropriate, systematic, and efficient mechanisms for health education and promotion, and in the case of rural schools they are often the social center of the community.

Fifth and seventh grade students were selected because children of this age have many of the basic skills needed to competently participate in an interactive curriculum. They are already the target of peer pressure and other social influences, and they represent two different developmental ages.

##### Project Aims

In September 1990, the Center for Indian Youth Program Development (within the Division of School



and Preventive Health, Department of Pediatrics, University of New Mexico) in partnership with Navajo and Pueblo communities and schools began a health promotion and cancer prevention project entitled "Pathways to Health," which was funded by the National Cancer Institute. The specific aims of this project are to:

- 1) develop, implement, and evaluate the efficacy of a health promotion program emphasizing cancer prevention strategies, specifically for fifth and seventh grade Navajo and Pueblo students, their families, and their schools;
- 2) design, implement, and evaluate with qualitative measures, a training program for school-based staff to effectively teach the curriculum and assure programmatic continuation; and
- 3) collect demographic, normative (knowledge, attitudes, and behaviors), and physical measurement data from this population with respect to selected chronic disease risk factors.

### Research Design

The research design is quasi-experimental with intervention sites (schools) randomly assigned to one of three conditions: curriculum only; curriculum plus family component; or control (delayed intervention with an alternative health education curriculum). The curriculum plus family component will be added to assigned schools in the third year of the project, and the control (delayed intervention) schools will receive the Pathways to Health curriculum in the final (fifth) year of the project. The development, implementation, and evaluation of the family component will be described in a future publication.

### Research Hypotheses

The research hypotheses of this project are:

- 1) students receiving the curriculum will show significantly more positive changes in knowledge, attitude, and behaviors related to curriculum content from pretest to post-test compared to students receiving the delayed intervention; and
- 2) students receiving the family component will show significantly more positive changes in knowledge, attitude, and behavior compared to students receiving the curriculum only.

### Curriculum Description

*This (Pathways to Health) project is based on the need to recognize and respect the traditions and cultures of the Indian people it serves. It builds upon centuries-old traditions, the living resources of the elders of the population, and native foods. It recognizes that changes have led to an epidemic of unhealthy lifestyles within the*

*population. It does not attempt to impose urban, middle-class values or lifestyles. The curriculum was developed to address the people in their respective environments, to educate the young to recognize and understand the consequences of unhealthy practices, and to teach preventive measures. (Ken Hunt, Center for Indian Youth Program Development, 1992)*

The Pathways to Health curriculum is a 16-lesson educational program designed to improve the health decision-making of fifth and seventh grade Navajo and Pueblo students through skills acquisition, self-discovery, and classroom discussion. The curriculum blends the values of healthful American Indian traditions with exploration of current health issues. Two primary behavioral goals are to:

- 1) promote a diet low in fat and high in fiber, fruits, and vegetables for the students and their families, and;
- 2) teach students to avoid cigarette smoking and the use of smokeless tobacco products.

Units within the curriculum include: Nutrition, Lifestyles, Tobacco, Social Pressures, and Advertising. Unique aspects of the Pathways to Health curriculum include intergenerational classroom presentations, student-initiated interviews with tribal elders, and the use of traditional and contemporary storytelling to convey curriculum messages. The variety of Native American contributions to the world's food supply is incorporated throughout the Nutrition Unit. The Tobacco Unit makes the distinction between ceremonial use and recreational abuse of tobacco, so that as opposed to commercial tobacco products, students understand that sacred, ceremonial tobacco is not to be avoided. Skills-building activities which assist students in resisting negative social influences (peer pressure, negative role models, and pressure from the media) are emphasized in the Social Pressures and Advertising Units.

A detailed process of curriculum development and testing included the use of focus groups of Navajo and Pueblo teachers and parents to identify key concepts and appropriate methods for school-based implementation and to assure reliability and sensitivity to cultural values. The project team which developed this curriculum is both multicultural and multidisciplinary. Teachers are trained to use the Pathways to Health curriculum during an "Educators' Training" session. In addition to an overview of the curriculum and its lesson activities, the educators' training includes presentations on comprehensive school health, intercultural communication, how this curriculum helps meet state educational (teaching) competencies, and curriculum evaluation procedures.

To promote implementation of the curriculum in the classroom, teachers are given the flexibility to augment the curriculum lesson plans with enrichment activities provided in the curriculum guide. They are also encouraged to customize lessons by contributing thematic community- and tribal-specific information, and to integrate activities from other subject areas. The intent of this flexible lesson plan is to enhance the learning experience of the students. Throughout the course of curriculum implementation, communication with teachers and school administrators is maintained through school visits, telephone calls, and project newsletters.

## PROGRAM EVALUATION

The Pathways to Health curriculum is being evaluated in several ways.

### Formative Evaluation

Formative evaluation activities occurred during the development and pilot testing phase of the project for modification and evaluation purposes. These activities included focus groups with school teachers, administrators, parents, and students to develop appropriate curriculum activities and implementation strategies. During the pilot implementation of the curriculum, teachers completed a brief evaluation of each lesson and provided qualitative information about their classroom's experience with the curriculum during an exit interview. Student response results from the curriculum testing instruments were analyzed for readability and for content validity via item analysis procedures. After each school visit, project staff completed a participant observer form to qualitatively record events that might enhance or deter program implementation. Minor revisions were made based on the combined results from the instruments, lesson evaluations, and teacher interviews. Data obtained from the pilot phase will not be aggregated with data collected after the curriculum was finalized.

### Process Evaluation

Process evaluation activities are currently underway to determine the extent of curriculum implementation in the classroom. Activities similar to those described in the formative evaluation section continue to be employed in this effort. In addition, the process of implementing and institutionalizing the program into the schools is being evaluated using the concerns-based approach developed by Gene E. Hall and Shirley M. Hord (*Change in Schools Facilitating the Process*). This approach provides a conceptual framework to evaluate the degree to which an innovation is incorporated into an existing program.

## Impact Evaluation

Pretesting and post-testing procedures are conducted prior to and shortly after completion of the classroom teaching. They are also conducted in the control schools during the same time period. Included in this testing are the completion of health knowledge, attitude, and behavior instruments by the students that reflect the curriculum content. Results of students' change in scores from pretest to post-test will help evaluate whether the program produced targeted changes in knowledge, attitude, and behavior in the curriculum participants. Standardized measurements of height and weight (for calculating Body Mass Index) and skinfold thickness at three sites (to estimate body fatness) are completed during pretesting only, to obtain descriptive information on this population. Body composition is not expected to change because of curriculum effects.

## PRELIMINARY DIETARY RESULTS

During the pilot test of this project, students were "pre-tested" on their existing knowledge, attitudes, and behaviors related to cancer, tobacco, nutrition, and other content covered in the Pathways to Health curriculum. Questionnaires were administered in a standardized procedure with each item read aloud twice to fifth graders and once to seventh graders. Three instruments were used to evaluate various aspects of food and nutrition. Specific food intake information was obtained on an instrument titled, "Yesterday's Food Choices." Results from this instrument and its validation procedures will be described in a future manuscript. General food frequency and nutrition knowledge information was obtained from "Youth Questions on Diet and Nutrition." Results from this questionnaire are reported elsewhere (18).

Specific nutrition knowledge related to the curriculum content was measured by a 19-item questionnaire. Two or three questions per Nutrition Unit lesson were asked on this instrument. Total score results from the administration of "Nutrition Knowledge Questions" are presented in Table 1. Mean scores for both fifth and

Table 1  
Results of Baseline Nutrition Knowledge - Total Score - by Grade\*

	5th Grade (N=158)	7th Grade (N=84)
Mean	9.7	11.9
Minimum	1.0	4.0
Maximum	17	18
Standard Deviation	3.5	3.5

\*A total of 19 questions



seventh graders indicate that they identified approximately half of the correct answers prior to being taught the curriculum. The range of scores was extreme, however, suggesting a wide range of nutrition knowledge and possibly of comprehension levels. The responses to certain questions provide insight into the students current understanding of the relationship of diet to cancer risk and the dietary fat and fiber content of commonly eaten foods. For example, when asked which "...person is at lowest risk of getting cancer?" only 41% of fifth graders and 57% of seventh graders correctly answered "someone who eats a lot of high-fiber foods" (Table 2). When students were asked to identify the type of milk with the least amount of fat, or the food that was the best source of fiber, only about half answered correctly. These results may indicate that students are unfamiliar with the fat content of skim milk, perhaps because of its infrequent availability in their communities (19). Also, while beans (burritos) are readily available, the knowledge that they are a good source of fiber is not widely known. For all responses, there were no significant differences between fifth and seventh grade scores.

## IMPLICATIONS

The Pathways to Health project aims to promote healthful lifestyle behavior in a culturally relevant way. The process of curriculum development, implementa-

tion, and evaluation described here can be helpful to those intending to foster healthful behaviors among members of other discrete populations. From the results of the nutrition knowledge questionnaire, it is clear that like many other children in the U.S. (20), the Navajo and Pueblo students who participated in the pilot phase of this project are lacking essential knowledge about the dietary fat and fiber content of key foods and the relationship of diet to cancer risk. Without this knowledge, targeted health attitudes and behaviors are unlikely to change.

## ACKNOWLEDGEMENTS

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(Continued on page 296)

Table 2

### Results of Baseline Nutrition Knowledge - Selected Responses - by Grade\*

	5th Grade (N=157) N (%)	7th Grade (N=84) N (%)
Person with lowest cancer risk?		
Smokes cigarettes	43 (27.6)	15 (18.3)
Eats high-fat foods	18 (11.5)	7 ( 8.5)
Uses chewing tobacco	31 (19.9)	13 (15.9)
Eats high-fiber foods*	64 (41.0)	47 (57.3)
Milk with least amount of fat?		
Whole milk	19 (12.1)	5 ( 6.0)
Skim milk	78 (49.7)	45 (53.6)
Low-fat milk	38 (24.2)	25 (29.8)
Milkshake	22 (14.0)	9 (10.7)
Best source of fiber?		
White bread	84 (53.5)	44 (52.4)
Bean burrito	30 (19.1)	21 (25.0)
Saltine cracker	23 (14.7)	12 (14.3)
Beef burrito	20 (12.7)	7 ( 8.3)

\*Underlined response is the correct answer

# CERVICAL CANCER PREVENTION

## AN INDIVIDUALIZED APPROACH

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### BACKGROUND

Until recent decades, the health status of American Indians and Alaska Natives (AIAN) has lagged far behind the rest of the population. Increased attention to this problem has produced positive changes, particularly since 1955, when responsibility for Indian health was transferred from the Bureau of Indian Affairs (BIA) to the Indian Health Service (IHS) of the United States Public Health Service (USPHS). Positive changes have come about in a three phase process. The first phase introduced improved environmental sanitation to traditional Indian homelands. These improvements provided immediate benefits with reduction in communicable diseases and infant mortality and with increased life span. The second phase began with introduction of chemotherapies for communicable diseases such as tuberculosis. The improvement in health status has been very successful where communicable diseases are concerned, but diseases associated with lifestyle remain to be addressed. Prevention and control of chronic diseases is now a major focus for Native American populations, just as it is for all other Americans. Therefore, the third and current phase of health improvement of Native Americans addresses lifestyle issues. Lifestyle issues for Native Americans are similar to those of the rest of the population. Important lifestyle issues include substance abuse, smoking, diet, stress, and early detection of diseases such as heart disease and cancer (1,2).

The health status of Native Americans has been improving but remains behind that of the U.S. as a whole. For the period 1980-1982, for example, the

mortality rate for Native Americans in areas served by the Indian Health Service was 778.3/100,000 or 1.4 times that of the U.S. as a whole. For females, the rate was 578.7/100,000 (3). Cancer rates among Native American populations are generally lower than those of other Americans, but the pattern of occurrence is distinct. Overall, the rate is approximately one-half to two-thirds the national overall rate for non-Indians living in the U.S. Based on data for 1980-1982 and using 1981 data on all races as a basis for comparison, cancer was the third leading cause of death behind heart diseases and accidents (98.4/100,000 for Native Americans, both sexes combined). For females, cancer was the second leading cause of death (diseases of the heart was first) at 89.4/100,000 (3). For Indians in the Nashville IHS area (the area that includes North Carolina), the age adjusted death rate from cancer in 1985 was 126.0/100,000, compared to 98.4/100,000 for all IHS areas and 131.6/100,000 for the U.S. all races. On the whole, Native Americans have lower rates of incidence of cancer of the lung, breast, and colon, but higher incidence rates for cancer of the gallbladder, kidney, and cervix. Native Americans also suffer higher mortality rates from cervical and gallbladder cancer (3).

There are few studies of cervical cancer among Native Americans and none focusing on those living in North Carolina. In two studies conducted in New Mexico, cervical carcinoma was found more frequently among Native American (7.65/1000) than among Caucasian women (5.82/1000) (4,5). At age 60 and older, the Native American cervical cancer rates were much higher than that of non-Indians (28/1000 vs. 12/1000) (4,5). Specific estimates for cervical cancer incidence and mortality among Native Americans are difficult to identify. Summary data are available from IHS service areas, but the data must be interpreted carefully because these service areas administer health care in more than one reservation of Indians. In the Nashville IHS service area (the area that includes the Eastern Band of the Cherokee), the 1985 age-adjusted mortality rate from

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cancer of the genital system for females was 14.7/100,000. The ratio of this rate to that of the U.S. as a whole is 1.1. In Robeson county, the traditional homeland of the Lumbee, the 1987 cervical cancer adjusted death rate was 4.78/100,000, compared to 4.62/100,000 for North Carolina as a whole.

AI/AN women have mortality rates from cervical cancer that are over twice as high as those for all women in the U.S. Between 1984 and 1988, the age-adjusted cervical cancer mortality rate for AI/AN women was 7.6 per 100,000, compared to 3.1 per 100,000 for all U.S. females (6). Identifying cervical cancer mortality among AI/AN females in North Carolina is difficult, but can be approximated using Indian Health Service data for the Nashville Area. During 1984-1988, the mortality rate from cervical cancer among AI/AN women in the Nashville IHS service area was 11.6 per 100,000. This mortality rate is nearly four times the rate for all U.S. females (6). The excess mortality from cervical cancer among AI/AN women is particularly troubling because cervical cancer can be detected at stages when treatment is effective in nearly all cases.

The North Carolina Native American Cervical Cancer Prevention Project (NCP) was developed to address the excess mortality from cervical cancer among AI/AN women in two tribes in North Carolina. With funding from the National Cancer Institute, this five-year project focused attention on preventing cervical cancer among Cherokee and Lumbee women. The Cherokee (Eastern Band of Cherokee Indians), which are a recognized tribe, reside on tribal lands in the western region of North Carolina and receive health care through the IHS at a service unit located in Cherokee, North Carolina. The largest concentration of the Lumbee tribe is in Robeson County, North Carolina. The Lumbee tribe is not Federally recognized, and tribal members are not eligible for health services through the IHS. Figure 1 identifies the target population within the State of North Carolina.

Table 1 presents descriptive statistics for the target populations, which include approximately 1500 Cherokee and 11,000 Lumbee women age 18 and older. As Table 1 shows, these women are relatively young (over half are under age 40), less than half have a high school education, and one-third are living below the poverty level.

### Need for community-based programs

The intervention for the NCP is a community-based, individualized health education program. The selection of a community-based approach was based on a desire to use a community model for intervention. The community model, as summarized by Weiss (7), incorporates the following concepts:

1. Interventions are provided in the environment where people live.
2. Community environments enhance the opportunities for information exchange and social support.
3. Using the community as a setting for interventions allows for testing of the efficacy of programs that is more generalizable than clinic-based trials.
4. Working in the community allows for observation of outcomes that are beyond the scope of the intervention and that may be related to public policy.
5. Community models allow for creative solutions to ethical dilemmas posed by withholding interventions.
6. The cost of community trials may be less than for individual trials because the intervention can be focused on groups.

For cancer control research in Native American populations, incorporation of the realities of daily living for individuals is exceedingly important. The barriers

faced by Native Americans in obtaining services needed for cancer prevention and control are barriers faced by the community and not by individuals alone. As a result, it was important that the NCP intervention reflect the realities of daily living in the target populations. Thus, a community based intervention was needed.

Previous experience with community-based cancer control interven-

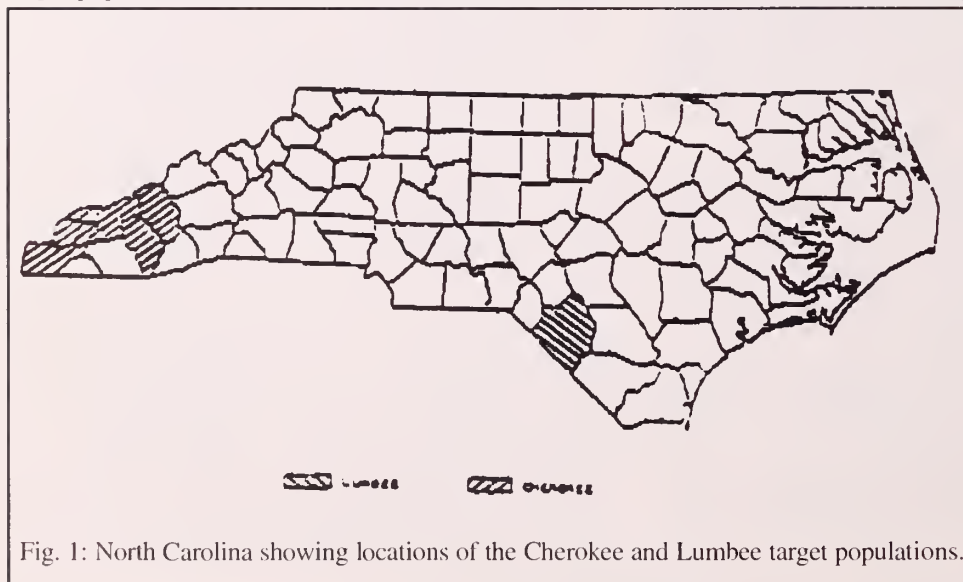


Fig. 1: North Carolina showing locations of the Cherokee and Lumbee target populations.

**Table 1**  
**Native American Women residing on Cherokee Tribal Lands and in**  
**Robeson County, North Carolina**

	Cherokee		Robeson Co.	
	Number	Percent	Number	Percent
Age				
20-29	443	31.3	3382	32.5
30-39	312	22.0	2397	23.0
40-49	240	16.9	1516	14.6
50-59	173	12.2	1431	13.7
60-69	143	10.1	1039	10.0
70+	106	7.5	697	6.2
<b>Total</b>	1417		10412	
Marital Status (Age 15+)				
Single	452	27.3	3342	27.0
Married	786	47.4	6480	52.3
Separated	92	5.5	648	5.2
Widowed	214	12.9	1347	10.9
Divorced	115	6.9	564	4.6
<b>Total</b>	1659		12381	
Education (years completed)*				
5-8	249	25.7	2352	33.5
9-11	315	32.6	2061	29.4
12	380	39.3	1995	28.4
16+	23	2.4	612	8.7
<b>Total</b>	967		7020	
Median years of Education				
*Females 25 and older	11.2		9.7	
Below Poverty Level (%)				
Native American Families		32.9		35.1
Female Headed Households		45.1		38.9

nity setting. The intervention would include instruction on cervical cancer prevention, information on how to gain access to screening and/or followup services in the community, and followup to address the barriers faced by individuals.

### Conceptual and Theoretical Bases for the Intervention

The design of the intervention for the NCP was based on Social Learning Theory (SLT) (13). This well-known theory presumes that most learning occurs as a result of a process of observational modeling, and furthermore that this process can be divided into four components: attention, retention, motor reproduction, and motivational processes. Compartmentalizing learning focuses intervention development on providing assessment, methods, and materials to address each part of the process.

A second theory that guided intervention development was self efficacy (14). Self efficacy is belief in the ability to behave in ways necessary to produce

tion strongly suggested that a mass media approach was unlikely to be successful in reaching project goals. The investigators had carried out a five-year cervical cancer prevention project using mass media in an urban area, and the results showed that the media was effective in increasing awareness of the issue of cervical cancer prevention, but that behavior change was not achieved (8-12). The NCP target populations are rural and access to mass media is not as certain as in urban areas. The lack of predictable access to media in the rural areas where the target populations live further discouraged the investigators from planning a mass media intervention. Most importantly, however, the behavior change achieved with the mass media approach was unimpressive.

The attractiveness of the community model added to the experience with limited effects from mass media interventions suggested that the intervention for the NCP should focus on the individual, but in a community setting. The result was an intervention designed to be provided to individual women, one-on-one, in a commu-

identified outcomes. This theory directs development of education involving active participation. Active participation is more effective in producing and maintaining behavior change than traditional "passive" educational methods (15). Several models were also used to structure intervention development and implementation. The Minority Health Communication Model (MHCM) was used to focus attention on the special issues related to developing health education for Native Americans (16). The MHCM divides the process of developing effective channels and methods of communication into five phases: consultation with "experts" on the minority group in question, consultation with "community representatives," setting goals, developing communication strategies, and evaluation.

To aid in identification of barriers to obtaining Pap smears and follow-up care, the PRECEDE (Predisposing, Reinforcing, and Enabling Causes in Educational Diagnosis and Evaluation) model was used (17). The PRECEDE model provided a sound conceptual basis for



planning community health education and directed consideration of the epidemiologic approach to analysis of social, cultural, and structural barriers to obtaining Pap smears.

Selection of educational methods, content and images, and means of communication was facilitated by the Communication-Behavior Change (CBC) Framework (See Figure 3) (18, 19). The CBC is a typology that links important elements of communication with the phases in behavior change processes, and in so doing guides the targeting of educational materials toward specific behavioral changes.

The integrative effect of these models provided (a) development of a comprehensive understanding of the target population in the context of the goals of the project, (b) selection of methods, and (c) development of educational materials that are appropriate for the needs, interests, and values of the Native American women in the target populations. The plan for development of educational materials and the modes of presentation used is summarized in Figure 2.

### Implementation of the Intervention

The overall goals of the project are to prevent cervical cancer by (a) increasing the proportion of women, age 18 and older, who receive Pap smears, and (b) increasing the proportion who receive followup care when needed. The intervention is delivered to women in the target population in face-to-face meetings. The purpose of the meetings is carefully explained and the women are offered documentation attesting to the legitimacy of the project, if necessary. Meetings usually take place in the women's homes and last from 30 to 60

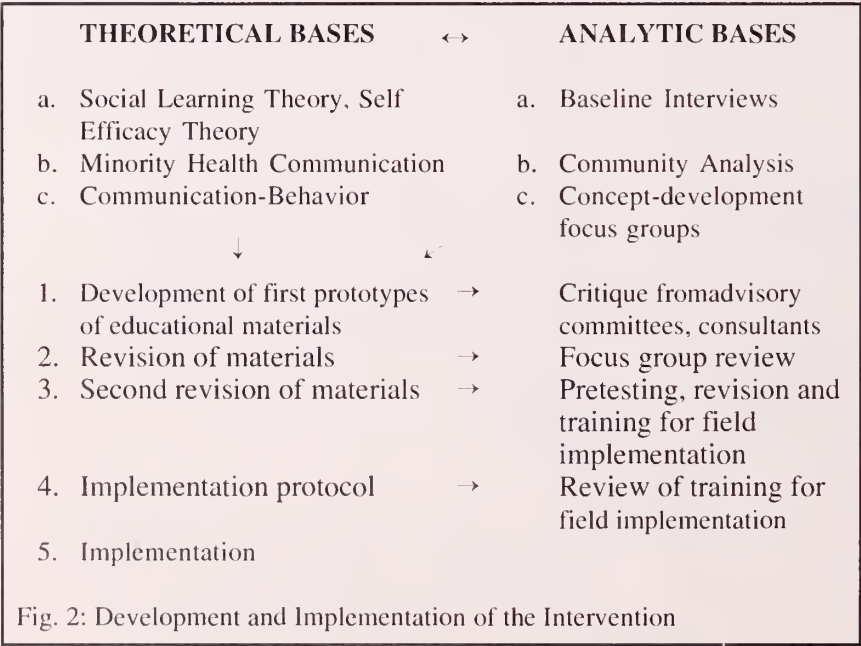
minutes. The basic concepts underlying the intervention are as follows:

- A. Cervical cancer, like most cancers, has the best chance for cure if found early.
- B. The Pap smear is the means for early detection of cervical cancer.
- C. Women need Pap smears throughout their adult lives. How often a women should have Pap smears depends on her own body. The doctor/nurse is the best source of information on how often to get Pap smears.
- D. Sometimes Pap smears find changes that indicate need for further care. If untreated, changes found by the Pap smear may lead to cancer.
- E. Obtaining further care when necessary is very important.

The means of communicating these concepts to the women includes a health risk appraisal, a 10-minute videotape, printed educational materials, and verbal interaction. The health risk appraisal provides a structured way to begin the intervention, provides information about overall health status and risk factors, and provides the intervention personnel a reason to schedule followup (to return the output from the health risk appraisal). The health risk appraisal (HRA) used, "Finding the Way: Health Risk Appraisal for Native Americans" (20,21), is an adapted version of a program provided through the Centers for Disease Control (CDC). The adaptations are focused on increasing the cultural relevance of the health risk appraisal for Native Americans in this target population.

A ten-minute videotape was developed by the investigators to provide structured information on cervical cancer prevention. The intervention is designed to be responsive to individual needs, but the flexibility leaves room for inadvertent omission of important factual information needed for the intervention. Showing the videotape to all women in the intervention ensures that all women will receive instruction on cervical cancer prevention at a predetermined level of intensity.

Printed educational materials include a pamphlet designed to accompany the videotape and additional materials on condyloma and followup care. The pamphlet to accompany the videotape is provided to all women in the in-



tervention, and additional materials are made available as determined by project personnel or as requested by women. Native American women from the Cherokee and Lumbee target populations have been recruited and trained to deliver the intervention.

## EVALUATION

Selection of a research design for the project was motivated by the principal goal of carrying out cancer control intervention evaluation while providing high quality risk reduction education for women in the target populations. In addition, the opportunity to use tribal rolls (exhaustive lists of all persons with tribal membership) to identify and select individuals for study made the use of a randomized design possible in an environment where individual residences are widely dispersed. Third, it was assumed that data collection would be carried out best in these populations by household interviews because of transportation barriers and lack of widespread telephone coverage. The Solomon Four Group design was selected for use (see Figure 3) because it allowed estimation of the desired effects, it controls for many threats to internal validity, and it can be implemented equally in both target populations (20). In addition the Solomon Four Group design allows for consideration of one of the most important factors affecting external validity by allowing for examination of testing effects and the interaction of testing and the intervention. Subjects were assigned at random to one of four groups for each tribe from eligible households. Thus, samples of 500 women from each tribe are included in the intervention groups and 500 are assigned to the control groups.

Evaluation data are collected in face-to-face interviews. The interviews are structured by a questionnaire to collect knowledge, attitudes, and behaviors (KAB) related to cervical cancer prevention.

Group	Sample Size (N)	Time 1	Intervention	Time 2
1	250	0	X	0
2	250	0		0
3	250		x	0
4	250			0

\*This design is replicated in the Cherokee and Lumbee target populations.

**Fig. 3: Evaluation Design\***

**Table 2**

**Selected Descriptive Data, Baseline Knowledge, Attitude and Behavior Interviews, Cherokee and Lumbee Populations**

Knowledge of the Pap smear*	(%)
Correct	74.6
Incorrect	7.1
Don't know	18.3
<b>Pap Smear Done During Last Pelvic Exam</b>	
Yes	84.7
No, Don't Know	15.3
<b>Most Recent Pap smear</b>	
Within past year	62.0
1-2 years ago	16.6
2-5 years ago	9.8
More than 5 years ago	7.6
Don't Know	4.0

\*knew what the Pap smear is and what it tests for

## Baseline Data Collection

The NCP is currently in the third year of operation. Baseline data have been collected and the intervention phase is underway. Table 2 summarizes selected data from the baseline interviews. As Table 2 shows, 62% of the women interviewed reported having had a Pap smear within the past year. From a conservative point of view, this could mean that nearly 40% of the women in the target population need Pap smears (assuming that these are high risk populations where Pap smears should be obtained annually). The proportion of women who correctly identified the Pap smear as a test for cervical cancer and who reported having had a Pap smear during their last pelvic examination suggests that the intervention should include teaching about the procedures involved with the Pap smear. Previous studies have shown that self reports of Pap smear history are often overestimates compared to the medical record (23). Medical record reviews are underway to validate the self reports provided on the KAB. Future reports on the NCP will provide data on agreement of the KAB and the medical records as well as on the efficacy of the intervention on increasing the proportion of women who obtain Pap smears.

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# HEALTH BELIEFS ABOUT CANCER AMONG THE LUISEÑO INDIANS OF CALIFORNIA

Diane Weiner<sup>(1)</sup>

## ABSTRACT

Among numerous Luiseño Indians of San Diego County, California, cancer is considered to be any tumor or growth that is possibly life threatening. A medical anthropology research project reveals that current and former Luiseño cancer patients share illness causation theories distinct from those of their kin. The patients believe their bouts with cancer stem from genetic predilection and/or God's will. In contrast, other Luiseño band members who do not have cancer assert that almost all cancer is caused by either 1.) chemical pollutants in the air, water, and food; or 2.) prior biomedical treatments that have gone awry. Imbalances of both natural and bodily elements are generally traced to particular culprits. This paper will detail these variances of Luiseño cancer etiologies and their impact on prevention policies.

## INTRODUCTION

The narratives of Luiseño cancer patients, their kin, and other band members illustrate the ways "... cultural values and social relationships shape how we perceive and monitor our bodies, label and categorize bodily symptoms, [and] interpret complaints in the particular context of our life situation. . . ." (1, p.xiii) This paper examines the variation among Luiseño Indian cancer etiologies and their impact on prevention policies. Clearly, health care knowledge varies intraculturally. Interestingly, current and former Luiseño cancer patients tend to share views of illness causation distinct from those theories of their kin and other band members. Of great importance is a commonly shared notion among the majority of Luiseño that cancer is a generally unpreventable condition. Conditions of direct and indirect experiences with cancer seem to greatly influence these beliefs (1-3). Involvement with biomedical and lay communication networks also appears to contribute to the health perceptions of a person (4).

## METHODOLOGY

Between June 1991 and October 1992, I undertook a medical anthropology dissertation field study on Luiseño chronic illness health practices. The goal of this project was to delineate and assess etiologies, methods of prevention and treatment of illnesses, and the utilization of biomedical and ethnomedical health resources.

Research participants included Luiseño band members from three reservations situated in northeastern San Diego County, California. Luiseño bands are currently situated on the La Jolla, Pauma, Pala, Rincon, and San Pasqual Reservations in San Diego County and on the Pechanga and Soboba Reservations in Riverside County. An Indian Health Service contract branch primary clinic is located on one of the reservations in San Diego County. As requested by members of the three separate communities, names of the reservations and of individuals as well as of other readily identifiable qualities of participants are excluded from this paper. All names used in this paper are pseudonyms.

Data collection took four forms: an epidemiological questionnaire, genagrams, formal interviews, and informal interviews. At least one member from each of 114 different households participated in an epidemiological survey. Participants from two reservations were randomly selected. Those interviewed from the third reservation were selected through opportunistic means because of limited access to reservation census data. The information collected from the questionnaire focused on the self reported prevalence of diabetes and other disabilities/chronic ailments.

During the second phase of research, four men and eleven women aided in the creation of genagrams. These kinship charts, based on family centered social work research practices (5), mapped the genealogies of eight individuals. Siblings and parents frequently assisted the focal narrator with genealogical information. The "ego" of each family chart then discussed her experiences as both vicarious and main participant in major illness episodes of family members. This work enabled me to document medical life histories, social structure, and health care support networks.

In addition to informal interviews with 51 individuals, 22 women and 11 men participated in intensive

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interviews concerning the perceived etiologies and methods of prevention and treatment of cancer, diabetes, asthma, and lupus. These individuals also described their utilization of health resources. More than half of the key research participants were interviewed on numerous occasions.

The sample population includes individuals between the ages of 18 and 91 years. All of the interview participants are enrolled members of a Luiseño band. Currently the different reservations are equivalent to distinct band memberships. Some of these individuals reside on reservations other than the ones in which they are enrolled. Others live off the reservation in rural areas or cities within a 25 mile radius.

## **WHO ARE THE LUISEÑO?: A HISTORICAL BACKGROUND**

A review of Luiseño history and culture is vital to the understanding of Luiseño perceptions of illness causation and prevention. According to 1991 reservation statistics, approximately 950 Luiseño inhabit the three reservations included in this study (6). Spouses and other affinal kin frequently reside in Luiseño households; householders who are not Luiseño are not included in these reservation census statistics. English is currently the primary language spoken on all of the Luiseño reservations; the native language, part of the Uto-Aztecan language family, is most often publicly spoken by those persons who are 60 years and older.

The Luiseño are the descendants of members of various semi-autonomous village political units that inhabited a region of approximately 1500 square miles. The aboriginal boundaries seem to have extended on the south from the Pacific Coast near Agua Hedionda Creek in what is now Carlsbad, to near Aliso Creek in the northwest near what is now Laguna. The eastward lines reached from Santiago Peak across to the eastern side of the Elsinore Fault Valley, south to Palomar Mountain and the valley of San Jose (7,8). The topography of this region includes a variety of ecological zones: coastal plains, grassy valleys, and foothills to the west; mountains and semi-arid niches to the north and east. Neighboring indigenous groups include the Kumeyaay to the south, the Diegueno to the south and the east, the Cupeno to the east, the Cahuilla to the north, and the Quechan to the southeast.

The traditional social structure of each village seems to have varied in accordance with population density (9). These semi-permanent villages were usually located in sheltered valleys near permanent water sources. Extended families and individuals, who formed villages, claimed communal rights to areas within band territories. Frequently, "groups of patrilineally related males" lived in a single cluster, and females either married out,

or married in, changing their residences to that of their husbands (10, p. 139). All lineages that shared a common ancestor formed a clan. In general, lineages from distinct clans joined together to establish "... endogamous territorial units that had political functions" (11, p. 15).

Patrilineages had exclusive rights to collect foods in a localized region within each village territory. Hunters were able to move throughout village lands in search of game (10,11). Village units also sometimes had rights of access to coastal lands and areas that contained other subsistence resources. Family groups often had household gardens and homes that were passed patrilineally and were maintained by wives and unmarried daughters (10, p. 62).

Semi-cultivated plants, such as grapes, acorns, and medicinals, were also harvested. Oak trees, whose acorns may have provided up to one ton yields (approximately 50% of inland Luiseño food supplies) were tended on communal lands. The acorns most commonly consumed were from the *Quercus kelloggii* and *Quercus agrifolia* varieties (10,12-13).

Leaders included both political and religious specialists. A male leader, who usually inherited his position, appears to have controlled both economic and warfare powers. This individual was assisted by a hierarchy consisting of an assistant, a village advisory council of elder males, and a secret society usually composed of all the younger men (10,12). Religious leaders also ideally inherited their positions and sacred powers patrilineally (10,11,13). Apparently a single religious supervisor was assisted by several ritual specialists, who each maintained relations with a particular mythic being.

Many Luiseño continue to view the period prior to contact with non-Indians as well as the era between the middle of the nineteenth and twentieth centuries as glory days, or as times of ideal access to Luiseño economic and cultural resources. Forced resettlement into missions (1776-1834), especially among those Luiseño who lived in low elevation western river valley areas, greatly disrupted the local economic and political structure (10,14). As European agricultural and livestock methods were introduced, lands for cultivation and grazing were enclosed. Access to ocean products was also curtailed.

During this period, many of the indigenous political leaders were prevented from employing their rights (11,15). Concurrently, as Catholicism was adopted by many individuals into the Luiseño culture, some Luiseño religious specialists began to practice covertly (16-18).

Between the years of mission secularization and Luiseño settlement upon U.S. federal trust lands (1834-1875), the Luiseño were ensconced in an era of socio-political and economic reorganization. Lowland and

upland Luiseño alike often realigned themselves into new political units incorporating the fragments and wholes of several distinct lineages. The political leadership generally encompassed "territorial chiefs," who were able to govern through power rather than consensus (11, p. 45). The native land bases shrank and became increasingly surrounded by private landholdings (12). Seven Luiseño Reservations: La Jolla, Pala, Pauma-Yuima, Pechanga, Rincon, San Pasqual, and Soboba were established by executive order between 1875 and 1892.

Two major events greatly impacted Luiseño control of band resources. In 1894, water was diverted from the San Luis Rey River to the Escondido Valley (19). In 1929, the U.S. government divided some of the reservations into allotments of 10 acres to heads of families and 5 acres to single persons, both adults and minors. The goal of the federal government as well as of many Luiseño proponents of allotment was to encourage family operated gardens and farms and to prevent further land loss.

Unfortunately, since the mid 1900s the diversion of water, periodic droughts, limited irrigation, and the restriction of lands have led to a decrease in horticultural activities, the gathering of wild plants, and the hunting of animals. Non-Indian operated and owned orange groves first began surrounding some of the valley reservations in 1931. Luiseño oral historians recount that World War II was the time in which a majority of band members first sought full-time permanent wage labor employment away from the reservations. A number of Luiseño elders also believe 1945 was the year in which drought conditions currently in existence first began. Limited rains and decreased water table levels have also impinged on the ability of Luiseño individuals to support subsistence and cash crop gardens and fruit and nut trees (13).

Throughout the 1950s and 1960s, many more Luiseño left their reservations to access employment, education, health, and social services resources. This exodus seems to have been partly inspired by federal termination policies and the transfer of federal government managed resources to California State jurisdiction. Reservation populations have greatly increased since the late 1970s. As one 71 year old man explains, "the programs have come back again. The people have come back to the blanket again; they went out, now they all want to come back to the res." Housing, education, and health services have all been re-introduced.

During the past decades about 4 - 5% of the allotments have passed into fee patent status. Non-Indian owned farmers and manufacturers have businesses on some of these parcels. One reservation has been home to a mushroom farm, a cabinet maker, a life jacket manufacturer and a trailer park. According to one Luiseño,

... We've had about 25 five acre parcels of land ... sold to non-Indians like the mushroom farm, and this other guy with toxic waste, whether the records indicate he's doing right, I don't know, but ... that's fee patented land that's been sold out. ... that mushroom farm is still under jurisdiction of the tribal council. We've got a moratorium right now; nobody can come in right now. ... [some Luiseño Reservations are] all tribal owned ... they don't have no allotments.

The tribal councils have replaced traditional Luiseño methods of leadership. Luiseño formal political structure is now based on articles of confederation that resemble the 1934 Indian Reorganization Act political institutions. The Luiseño band government system originated in 1953, when reservation residents wrote articles of confederation and established formal reservation membership requirements. Each reservation has elected officials who preside over reservation business and general community meetings. The objective of these meetings is to aid all adult band members to reach consensus on community issues.

The current reservation residents frequently embrace tenets of native, Catholic, and Protestant ideologies. Most Luiseño religious ideas and practices remain private. Since the late 1970s, a minority of individuals have attended evangelical church services. It is not uncommon for Protestants to decrease participation in religion after a few years. In general, both Catholics and Protestants tend to integrate and/or add these belief systems to previously adopted doctrines.

## CONTEMPORARY HEALTH SYSTEMS

Although no individual Luiseño is currently formally recognized as having divine healing capabilities, certain men and women are noted to be knowledgeable about herbal remedies and other ethnomedical practices. Historically, male healers derived their secret powers through dreams and contact with sacred cosmic beings. Male and female herbalists, masseurs, and diagnosticians also served the ailing.

Well-being exists when individuals and groups maintain Luiseño rules of reciprocity, respect, and unity (16,20-22). Illness among the Luiseño was reportedly caused by accidents, sorcery, transgression of social rules and/or the innate inability of an initiate to complete a particular ceremony (20,21,23-25). Treatment methods varied in accordance with illness etiologies (20,21).

Currently Luiseño individuals have a plethora of treatment options from which to choose. Even though the overwhelming majority of people with whom I had contact utilize biomedical providers associated with an



Indian Health Service primary care contract facility, many persons also seek the care of practitioners affiliated with health maintenance organizations, veterans association hospitals, urgent care units, and private medical practices. All but one of the cancer patients interviewed reportedly used physicians referred by the Indian Health Service facility providers. The Indian Health Service branch does not employ these physicians.

Often people obtain ethnomedical assistance in the forms of massage, herbal remedies, counseling, and private family ceremonies. Prayers—Luiseño, Catholic, and Protestant—as well as the laying on of hands are also used to relieve pain and suffering associated with illness. One local biomedical provider maintains many of his clients continue to adhere to the belief that social and sacred transgressions cause emotional and physical distress.

## **THE INCIDENCE OF CANCER AMONG THE LUISEÑO**

Many Luiseño individuals assert that cancer is a common ailment among band members. Malignant neoplasms are currently the third leading cause of death among American Indians nationwide (31). Of the few existing studies concerning American Indian cancer incidence and mortality, distinct and varied methodologies and time frames are often used. American Indian ethnicity is often misrepresented or unreported in tumor registries (26,27). Indeed, “until recently cancer has not been acknowledged as a major health problem for American Indians” (28, p. 25). However, incidences of cancer seem to be increasing in correlation with the American Indian life expectancy rate. American Indian rates of cancer seem to be also impacted by dietary changes and by alcohol and tobacco consumption. It is also possible that more native individuals are being diagnosed than in prior periods (28,29).

Self-reported cancer incidence among the Luiseño seems to reflect a diversity concerning what types of ailments are viewed as disabling. Some of those people surveyed list cancer as a disability; others mention conditions of cancer while discussing other past or current medical problems. Members of 10 of 114 Luiseño households surveyed reported a current cancer patient or individual who claims to be in remission. Five of the individuals also claim to have had diabetes at the time of cancer onset.

Among those surveyed, types of cancer include the cervix, breast, kidneys, lung, ovary, retina, and multiple myeloma. Two males and three females are reported to have had (or had) bouts with colon cancer. Colon cancer is the second leading cause of American Indian cancer deaths (30). Surprisingly, though, mortality rates for

colon cancer among California Indians are lower than those of other Californians (31,32). Luiseño females claim to suffer from kidney and lung cancer. The California Indian cancer mortality rates for these cancer sites are below the national average among the general population and American Indians nationwide (31). California Indian mortality rates for retinal blastoma are reportedly negligible (31). All of the figures on California Indians must be viewed cautiously. According to the most recent research conducted by California Indian Health Service personnel, widespread underreporting and misrepresentation of California American Indian cancer diagnoses and ethnic classification often occurs (Yow, personal communication, September 1992).

## **LUISEÑO VIEWS ON CANCER**

According to many Luiseño individuals, cancer is perceived to be any tumor or growth that is possibly life threatening. People differentiate between types of cancer: distinctions are drawn in relation to which organ is first diagnosed with the disease. Those interviewed feel cancer varies because it can attack any part of your system. Apparently if only one organ is affected, the name of the cancer is associated with that organ. If more than one part of the body is impacted, people will refer to the problem in total as cancer or as the type of cancer first diagnosed. For instance, a person with uterine cancer who develops lymph node cancer would often be described as having uterine cancer.

All research participants agree that cancer is also considered to be a relatively new condition — one which became more predominant only in the early 1970s. As one person declares, “a long time ago we didn’t know these things. . . . we didn’t hear about people dying of cancer, at least we didn’t know about it. Not like all the people now do.” Even though both those with direct and indirect experiences with cancer concur that diagnoses among the Luiseño are gaining frequency, strong distinctions concerning the descriptions, causes, and means of prevention are evident.

## **INDIRECT EXPERIENCES WITH CANCER**

Among those Luiseño individuals who have not had cancer of any kind, cancer is generally described as “a runaway growth” that starts and “spreads through the system.” These growths may be referred to as “boils,” “cysts,” “tumors,” or “warts.” Cancer is “like a fungus or something, it eats you away.” This last description is extremely similar to that used by urban Anglo working class individuals and rural Anishinaabe people (33,34, p. 161).

All cancers are associated with “pain, misery, and agony.” This suffering stems partially from the ability

of these tumors and growths to “hide, travel,” and “hit on things” in the body. As Sontag has written, “metaphorically, cancer is not so much a disease of time as a disease or pathology of space” (35, p. 14).

Among the Luiseño, cancer seems to be a visual acknowledgement and representation of both an imbalanced body and society. A “weak” or “broken down” body and its immune system is reportedly extremely susceptible to “a build up” of cancer, a condition that not only signifies, but accentuates disharmony. Bodies and blood that are “clean” are in balance and are apparently less susceptible to impurities such as cancer. This concept is more easily understood in the context of illness causation theories.

Myriad Luiseño people who have had indirect contact with cancer state this ailment has multiple etiologies and that it is often difficult to pinpoint one cause (34). Some individuals assert that cancer has mysterious origins; however, these people also tend to think tumors and growths “are triggered” or develop due to contact with invasive impurities. One woman outlines the views of many other Luiseño in her description of the relation between cancer onset and contact with toxins:

Problems like cancer [exist] cause we’re out of balance and no longer using herbal remedies; we don’t live off of the earth. . . . I think the world is cancer giving because of all the chemicals that are used, even as far as fruit that we grow . . . they have the insecticides. The cans they use to store their fruit and just about anything they process, as far as manufacturing the product and all that — it’s all toxin, you know. It’s not like in the old days when the ground was virgin land and it all was good soil and vitamins and minerals in the ground and you come out with nice vegetables, health stuff. Cause back then nobody got Cancer; think about it, nobody got Cancer back then, a long time ago.

[The grove owners have] been doing spraying quite a bit and they’ve been coming pretty low; in fact we have so much air space right here . . . and nobody’s looked into it and these planes are coming lower, lower than these tree tops, these eucalyptus tree tops. . . . I could taste the infection . . . and the wind blows and the trees are all around us. See, that stuff they spray down there, and then the oranges go to market and they got that insecticide on them, even our oranges are contaminated . . . the insecticide is doing something to our lungs; it’s dry and you gotta cough and it’s scratchy . . . I know they’re invading our air space; they’ve never come this

low . . . we’ve got groves on both sides of our reservation.

In addition to contact with food preservatives, insecticides, and pesticides, research participants list a variety of visible regional sources of possible air, water, and soil contamination. Contaminants reportedly stem from the cabinet, mushroom, and life jacket manufacturers. According to one woman, there is “environmental pollution from [the life jacket manufacturer] or the wood factory solution on the boards. There are weird fumes around here. The mushroom factory always smells bad.”

The San Diego County Toxin Release Inventory claims no toxins have been registered as being released in this or in the surrounding zip code areas. Nevertheless, other Luiseño individuals believe substances associated with military testing at the Chocolate Mountains Marine Base and air borne emissions from San Diego County factories and automobile traffic increase Luiseño susceptibility to cancer. Native Alaskan informants discussing cancer etiologies also mention nuclear toxins and air borne emissions (36).

The Luiseño people who feel cancer may be associated with these types of contaminants assume band council members and non-Indian businessmen and governmental officials are partially responsible for cancer onset. As one Luiseño person states, non-Indian “society came in and changed the belief system....we’re destroying ourselves because of the ways societies govern us.” Non-Indian business and government officials are classified as “invaders,” who allow and/or promote the use of these toxins (See 33, p. 434 for comparable attitudes among the Anishinaabeg).

Luiseño governing bodies of the past century may be seen as culpable since they acquiesced to non-Indian government policies of allotment and fee-patent land-holdings that have passed into the control of non-Indian manufacturers and agribusinesses. Past and present leaders reportedly have not aggressively pursued either Luiseño rights to land loss or the protection of air space and water. Most fascinating is that a number of the people who make these assertions are or have been involved in local government or are the children and grandchildren of these leaders. Apparently, responsibility thus encircles all band members. As Black Feather (37, p. 140) observes in a discussion of the group dynamic among Native Americans:

Even today, Indians seldom, if ever talk about a given individual’s success, but they might say that the tribal group has enjoyed success . . . the Indian community strives for interdependence, collective responsibility, and cooperation through a definitive hierarchy.



Blame for and obligation to assuage a problem rests among leaders as well as those who selected these officials. Band council members are elected by the general populace over the age of 21 years. The tale of one former band council member who was working to resolve a different issue clarifies this notion of responsibility:

... they told me to go do something, and I failed miserably ... and the band, some of the people were crucifying me — calling me names — this old man got up and he said, “three years ago we told this man to do something, you, you, and you — you told him to do something. ... He did it. So it went wrong, something went wrong. ... But if he failed, I failed too ... if you’re scolding him, then you’re gonna have to scold me too.”

Collective responsibility of communal problems contrast with perceived individual duty to maintain well-being. Some substances, including tobacco, alcohol, and illicit drugs, are also thought to be cancer causing. The ingestion of these items is directly associated with the onset of lung, pancreatic, and vaginal cancers, respectively. Luiseño individuals also claim contaminants associated with certain household cleansers and other “man made chemicals” are “on the long run things you gotta watch out for.” These products as well as processed food items, such as “the bleached flour and lard we use in our tortillas,” “pepsi,” and beef appear to be associated with a variety of cancers. As one man puts it, “refined stuff in big doses plugs up your system, probably causing cancer.” People who ingest these items are sometimes thought to be personally responsible for later illness effects.

Another woman explains, “I think a lot of people who die of cancer didn’t do anything to bring it on.” When asked if some people bring on their illnesses, this woman, a cigarette smoker, responded that

Lots of people don’t work in factories, or smoke, they live a decent life, maybe they didn’t eat greens ... when they were younger. ... I think it’s all this stuff in the air and I don’t know what it all is ... or there’s cancer in all of us and [something] gets it moving.

A few other women agree that “we all carry the germ for cancer ... for some people it starts moving and for others it never does.” Two Luiseño educators and one nurse feel psychological, spiritual, and physiological stress may induce cancer. They too feel cancer “germs” may be in all people.

Another way cancer is thought to occur and spread is through direct bodily impact. Bruises to the body report-

edly may cause cancer to the part damaged. Biomedical interventions also are felt to be pathways of danger. One individual attributed the deaths of two extended family members to surgical procedures.

Cancer feeds on incisions. ... or openings up or something. See what happened to my aunt, they went in and did [gallstone] surgery on her. ... they did surgery on her, and she was recovering fine but the only thing that happened was the first week after they did the surgery ... she was doing ok and then the second week, she was losing her weight. So they went back in and [cancer] was hiding behind her bladder. So they went back in once they diagnosed it and they thought they got it all, but by then I guess from being opened up so much, so many times it had spread.

I heard that it hits on (pause) air, like when they open you up ... it seems once they diagnose somebody and they go in and try and take it out, the people end up having more cancer, or it’s travelled. I don’t know how it travels or how fast it travels. I don’t know if the doctors really know.

This notion that medical interventions may induce cancer or promote its spread is not shared by individuals who have had cancer. Those persons who have had direct experience with this illness attribute their situations to different causes.

## DIRECT CONTACT WITH CANCER

Luiseño individuals who have had experiences with cancer describe this health condition differently from their kin. All but one Luiseño cancer patient with whom I spoke detail their narratives of cancer with both biomedical jargon and metaphorical qualities. One woman explained she is “short winded” and in “pain but not too much” because she has ovarian and stomach cancer, with “small nodules gone to my lungs.” These people all distinguish benign tumors and cysts from malignancies that signify cancerous cells in the body. One woman, Martha, said,

Cancer, I think maybe it’s a sore, that it starts out as a sore and just consumes. ... the body or whatever area it affected. It just kinda grows ... when I think of cancer I think of the blob, remember that movie? And how, you know when they were running from it and it just came up under the door, and just plops all over. It may begin small but just kind up eats up everything it touches.

Additionally, when asked to explain “what is cancer” and “what causes cancer” each person described their personal experiences beginning with symptomatic onset of the condition. The following discourse from Jake, who claims to be in remission from colon cancer, seems representative of most of those interviewed:

I didn’t know I had cancer ‘til they told me. I went to the clinic, the Indian clinic [one Friday]. See, I was losing blood in the colon (ID: oh). I was sick but I didn’t say nothing, I didn’t know what was wrong with me. I’d run out of breath. I’d come out here [to the yard] and try to shift the gate, and I couldn’t even get up the stairs, you know. So I get up there [to the clinic]. — that nurse up there. . . . she came down and looked at me.

“My gosh” she says, “you’re not right, you’re not looking well. We’ve got to get you to the hospital.”

“O.K.,” I say. “Maybe I can go on Monday.”

“No,” she says. “We got to get you there now.”

So by golly they sent me down, wrote me a ticket that said what was wrong: low blood count. . . . I went right into the ER. They said “we got to keep you here, so they operated. . . .”

Current and former cancer patients also tend to describe cancer as multi-etiological. Nevertheless, each cancer patient separates etiologies of his/her illness episodes from those of other people. These cancer patients assert that the causes of cancer for non-Indians often remain mysterious. This disease is thought to arise among Indians in general due to lifestyle habits, including the consumption of tobacco and alcohol, “fatty” foods, and/or exposure to local environmental and chemical pollutants. However, cancer patients themselves have more idiosyncratic beliefs as to what causes cancer.

A number of cancer patients believe that they have a genetic predilection to this ailment. For instance, Martha traces the onset of her grandmother’s lung cancer to her cigarette smoking habits. Yet Martha states, “for myself, I would think perhaps something in my genetic make-up, or something, perhaps something [in my cells] didn’t match so cancer started to grow.” Martha also believes her genetic condition may have possibly been impacted by unknown environmental or life style habits of her biological mother who has recently been diagnosed with cancer of the stomach.

Several individuals also believe their illnesses may

be traced to God’s will. God apparently does not necessarily cause cancer — this problem seems to stem from malevolent cosmological forces. A few women have traced historical and contemporary causes of illnesses and troubles to the social transgressions of other Luiseño individuals. These violations, of which sorcery is one, jeopardize individual and collective existence. Unintentional and intentional religious or social infractions may also influence the health of the transgressor and/or family members (16,20-22,24,38,39).

Historically, Luiseño sorcerers were thought to have been able to send illness into the body through intrusive objects — and the removal of these invasive items is part of the healing process (9,20,21,24). It is possible, although not confirmed, that cancerous growths and tumors that “hit on you” are considered to be related to such intrusive objects (40). In the past, malevolent acts were frequently associated with bodily intrusions and untreatable conditions. Diseases of an epidemic nature, including tuberculosis, measles, and smallpox, seem to have also been attributed to the malevolent practices of individuals (8,21). An aunt of one woman once accused her of being especially evil and cruel as a child, thus instigating the cancer in herself and in her relatives.

Some of the women who attribute cancer causation to evil beings often attend either Catholic or Protestant church services and read the Bible daily. Beth, 24 years old, who is currently in remission from cervical cancer, frequently includes Catholic, Protestant, and Luiseño religious reasonings and metaphors in her discourse. She states,

. . . bad things come from Satan and I think all good comes from God . . . from Satan comes death. . . . He comes to kill, steal, and destroy, and I believe that. A lot of times my mom will say, well. . . Indians have a lot of superstitions, ways, and tell-tale stories, but [my mom] always says it was witchcraft . . . I’ve even had tricks and things played on me and still do sometimes, and it’s like, oh well, I just brush it off and say “God is my protector.”

. . . I know God’s a healer and I know God’s here, and how do I put this into my own head that he’s here and he’s healing me and going to . . . cure me of cancer?

Unlike his counterparts, Jake does not attribute his cancer occurrence to genetic or divine sources. He ascribes his situation to only one etiology. Indeed, Jake states he was informed by his physicians that his colon cancer was a result of his eating habits. By eating “mostly greasy stuff, and fried foods . . . the food that you take in” does not allow the intestines to be “cleaned out.” Thus, according to Jake and his physicians, colon cancer may ensue.



## THE DIVERSITY OF KNOWLEDGE BASES

Jake is quite clear about the source of his health care information and also the foundation for his beliefs. An assessment of the variables that seem to influence the illness causation ideas of other Luiseño people is imperative for comprehending Luiseño views of cancer prevention. In addition to direct versus indirect experience with cancer, access to medical research and kinship networks seems to greatly influence these cancer etiologies.

As revealed by their descriptions, Luiseño cancer patients for the most part have the ability to understand and recreate biomedical codes of meaning. The language of patients tends to be framed by both common-sense cultural knowledge and biomedical jargon (41). The personalization of illness events decreases and/or limits the objectification of the relationship between facts and values, physical manifestations and social contexts, and body and self (42-44).

The perceptions of Luiseño cancer patients are frequently based on interactions with biomedical personnel and other cancer patients (2,3,45). Even those individuals who attribute cancer to cosmological forces assert that much of their knowledge about the effects of cancer are based on dialogues with physicians, nutritionists, and other family members who have been cancer clients. On several occasions, Luiseño cancer patients have reiterated that they are the type of people who ask questions of their providers and who actively seek comprehensible answers to these inquiries. Some of the cancer patients attempt to clarify and extend their knowledge of health through radio and literary resources.

Only one woman, Claudia, who claims she does not understand what causes cancer, does not seem to rely upon biomedical practitioners as information resources. Claudia told me she refuses to look at her surgical scars and does not know if after two surgeries in 1992 whether or not she has cancer. It is unclear to me whether Claudia is unwilling or unable to interpret biomedical codes. She, like one woman who reports having had a benign tumor removed, may feel that to observe and to discuss her ailment implies self condemnation. For Claudia, acknowledgement of cancer, rather than merely of a "tumor" *per se*, may represent defeat and impending disaster (46).

Three months after her second surgery, Claudia informed me that she had a cyst the size of a volley ball removed from her abdominal area. She sprinkled her account with a few biomedical terms through which she explained why her *cyst* caused such discomfort. And, unlike our prior meetings, "death talk" and conversations about God never occurred.

Those who have had only secondary experiences with this illness rarely state that their etiologies are

based on direct contact with biomedical personnel. Instead, knowledge is generally based on second and third hand sources of information. In her discussion of an aunt's demise, one woman relates, "I remember someone saying it's in her lymph nodes and that's why she had so much pain."

Individuals who feel cancer is stress induced claim to have read such information in educational or health research reports. A number of persons who link cancer to environmental factors and/or to food, alcohol, and tobacco intake cite their sources of information as radio programs, books, and news articles. Frequently these individuals also construct their ideas based on visible changes to the local physical and cultural environments. As one man states,

Let's see . . . I listen to this one station on this radio show sometimes. A nutritionist — I get some of my information that way, whether it's true or not. . . . A lot of this, like I hear and put in my own two cents, you read it in books and you know it says this, so this must be happening too . . . whether it's true or not, I try to use my own judgement.

Daily or weekly communication between siblings, parents and children, and aunts, uncles, nieces, and nephews also seems to influence perceptions about these etiologies. Even though individuals have many idiosyncratic beliefs, some kin groups tend to have cluster etiologies. This situation may reflect verbal and nonverbal mechanisms of sharing cultural medical knowledge (4,47,48). For instance, both an uncle and niece who live less than a mile apart focus their beliefs on the cleanliness of blood and organs. Family members of another three households who have constant daily interactions with one another all emphasize that chemical pollutants cause cancer. These people also claim to rely on one another as well as upon media resources for health care information. Such communication practices also extend to other kin clusters.

## UISEÑO METHODS OF CANCER PREVENTION: SECONDHANDEXPERIENCES

Luiseño sources of health knowledge seem almost inseparable from notions of prevention. The majority of individuals who have not directly experienced cancer believe this disease to be largely unpreventable and untreatable. Individuals do however, have some resources and responsibilities for the care of their bodies. A few women state that annual breast and gynecological exams may at least detect cancers in their early states thus possibly avoiding amputations and/or surgeries.

One can also apparently maintain bodily harmony

through a variety of means. According to research participants, one should "drink a lot of water, I say in my mind, whether it's true or not, with all this water I'm drinking I don't think I have time to get any kind of something, cause I'm cleansing it all times." Individuals believe they should also exercise, refrain from the use of alcohol, tobacco, and illicit drugs, and limit the ingestion of household cleansers. Some people suggest that individuals "need a balance, a balance of greens" in their diets. The consumption of vegetables "straight from the ground, fresh" or "frozen" that supply Vitamins A, B, C, and fiber is reportedly beneficial. Limiting the intake of lard and other fats is also considered necessary.

Reservation horticultural activities consist of garden produced supplements to diets. Since almost all Luiseño currently rely on store bought or commodity distributed food items, it is deemed difficult to control the ingestion of cancer causing chemicals. People also feel that unless toxins released on and around the reservations cease, anyone can be a cancer target. Only if Luiseño individuals can influence environmental and legal policies that impact reservation and regional health conditions will cancer have less opportunity to strike (34, p. 165). The attitude that the onset of cancer is almost uncontrollable is also shared by those persons who believe this disease has mysterious origins.

Perhaps most importantly, all of these individuals agree that cancer is basically untreatable. Medical interventions such as chemotherapy and "cobalt treatments just prolong problems." These procedures are thought to merely add months or years of pain and agony to the life of cancer patients. Those who perceive that biomedical interventions are detrimental to the body believe that invasive procedures including amputations should be avoided.

## **LUISEÑO METHODS OF CANCER PREVENTION: FIRSTHAND EXPERIENCES**

People who have had cancer feel that although it may strike anyone, this health condition may also be preventable. For those who may have a genetic predilection to cancer of any kind, individuals feel one should "go back now and again" for physical examinations. Martha has told me on numerous occasions that genetic, prenatal, and postnatal counseling would be extremely helpful for prospective parent(s) who believe they may "carry the cells" of cancer. Such counseling may aid individuals to make choices that would be socially and psychologically appropriate for the possible parent(s). In addition, she associates certain cancers with prior radiation therapies:

Like say anybody that has special kinds of treatment, or something the body wouldn't ordinarily be exposed to, just kind of watch for

different kinds of things. Last August I found out I had a brain tumor but I think it may not have been related to the retinal blastoma because if it had been, then [the tumor] would have been malignant. . . . The doctor thought it could have been from the radiation. . . . I had radiation when I was about 18 months old and...my birth mother says I was in the hospital for the first two and 1/2 years of my life the whole time. . . . she said the radiation had been so strong that it burnt my face so severely. . . . Be aware of what you're doing to your body. To fight one thing might cause something else.

Another way to possibly prevent malignancies is through the maintenance of a diet, especially one that is developed by a nutritionist or physician. This assumption is shared by Jake as well as by those who feel cancer may be caused by divine/supernatural events. Individuals feel all Luiseño should restrict the intake of caffeine and "fatty, fried, and greasy foods." People who adhere to these beliefs argue that it is not necessary to eliminate items such as coffee, sweets, and beef from the diet, but rather to decrease their consumption and to increase the consumption of fibrous grains and vegetables. All those interviewed also claim that they have not used alcohol or cigarettes since their diagnoses with cancer.

According to cancer patients, if this illness is not always preventable, it is generally treatable. All those with whom I spoke claimed they delayed seeking care when first experiencing symptoms. Many individuals did not associate their pains or other symptoms with cancer. Cancer diagnoses and treatment almost always ensued because symptoms prevented the person from maintaining daily activities, and then a spouse, parent, child, or sibling, insisted they obtain immediate care (40).

Health care providers who have assisted American Indian cancer patients note that patients often take "ownership" of the disease and strive to maintain biomedical treatment regimens (37, p. 143). The narratives of Luiseño cancer patients tend to demonstrate patient empowerment. Their accounts stress decisions and choices made. These judgments and actions were and continue to be pursued in cooperation with others: kin, biomedical personnel, and ethnomedical practitioners. All but Beth claim that they regularly attended or currently attend cancer treatment therapies and follow-up care. They also often use ethnomedical therapies as well. Beth, who again tested positive for cervical cancer three years after her first "kryo" surgery, refused any medical care for several months. During this interval, she told me,

Not that I didn't have faith in God . . . but I don't want to test positive again . . . they're



talking about doing a hysterectomy, and they want to send me to specialists and all that, no way . . . not that I don't have faith in God . . . that's why it's such a big, a let down on my part. Sometimes I feel like I'm failing God because I don't believe in . . . faith healing. . . .

A few months later when we spoke, she said her husband had threatened to carry her into the car and drive her kicking and screaming to the office of her physician. She assented to further testing, which resulted in a negative diagnosis. Beth now claims she will combine biomedical and faith healing therapies with a regimen of exercise and diet as prescribed by the local Indian Health Service nutritionist.

The use of plural medical systems by cancer patients may represent a belief that biomedical care might alter and alleviate symptoms, but ethnomedical practices may aid and/or heal the spirit. Herbal remedies, prayer, counseling, and other forms of healing may be considered crucial avenues towards wellness (39,45). Yet, it is highly likely that the use of such health resources will not be discussed by patients and biomedical health care workers.

Beth, as well as other cancer clients, clearly want control over their bodies. Many Luiseño indicate interaction with biomedical personnel represents the actuality of the disease. In contrast to their kin who have not experienced cancer, these individuals seem to need a period of internal negotiation in which medical therapies are deemed appropriate. This period of time may vary from one individual to the next in accordance with other illness experiences, age, and cultural views. This negotiation may also be impacted by a patient's view of his/her household nurturing role. Those individuals who feel other people rely on their well-being for daily needs may address health care practices in ways that expedite treatments.

Once this internal mediation is resolved, a diagnosis of cancer is not usually viewed as a death note. Biomedical and often ethnomedical care thus become possible methods to regain some control of the body. Hope and treatments thus seem to become synonymous. For Luiseño cancer patients, choices concerning care include the assistance of select kin and health providers. Unlike cancer patients in the dominant society, Luiseño patients do not "will cancer away" (36,48). Instead, individuals tend to rely upon available social, spiritual, and technological resources in order to "slow down the process" of cancer "spreading." A collection of resources appears to be viewed as the ideal means of combatting this indiscriminating illness.

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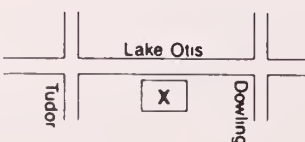
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# HELPING THE NATIVE AMERICAN CANCER PATIENT, THEIR FAMILY AND COMMUNITY

## A Panel Presentation

Judith Salmon Kaur, M.D.

### ABSTRACT

**A panel presentation comprising two Native American cancer survivors and the widow of a cancer patient discussed some of the personal and cultural issues faced by the Native American cancer patient.**

In June 1989, North Dakota initiated a new Cancer Prevention and Control Program funded through the North Dakota State Department of Health under a grant from the National Cancer Institute (NCI). The twelve member coalition formed to assess health needs and problems related to cancer included myself as medical oncology consultant, Dr. Thomas Welty, epidemiologist from the Aberdeen office of the Indian Health Service, the Executive Vice-president of the American Cancer Society, and representatives from a number of other organizations involved in cancer control and prevention. A Native American subcommittee was formed from this coalition to set up an intervention project on the Fort Berthold Reservation in North Dakota.

Hampton has previously pointed out the heterogeneity of cancer in Native American populations (1). As the coalition reviewed data from the Indian Health Service from 1968-1988, they found that regional differences in cancer incidence were striking (2). In the Aberdeen area, for example, cervical cancer is a significant cause of mortality for Native American women with a rate that is greater than four times the national average (Fig. 1). Because of that data and the documented efficacy of screening and early intervention, cervical cancer screening therefore became a high priority in this demonstration project. Recognizing a need for culturally acceptable materials, the NCI utilized Dr. Kaur and Native women from North Dakota to produce a teaching videotape on Pap smears and cervical cancer to be used as part of an educational program to reduce the mortality rate of cervical cancer in this population.

In another example of differences in cancer mortality, the coalition found — as shown in Figure 2 — that breast cancer mortality is lower in Native American women (14.6 per 100,000) compared to the U.S. all races rate (27.3 per 100,000) (2). In general, however, mortal-

ity rates for most Native American patients with cancer are higher than in the non-Indian population.

Survival data recorded by SEER (Surveillance, Epidemiology, and End Results) for different races showed that in general survival in Native Americans from cancer at one, three, and five years is the poorest of any group studied (3). Only by understanding the wide divergence of types and frequency of malignant neoplasms and developing tribal-based interventions can this terrible pattern be changed. Emphasis must be placed on prevention, screening, and accessing state-of-the-art care. In addition, better supportive care in the home community can make treatments more humane for patients.

At the First National Conference on Cancer in Native Americans held in 1989 in Tucson, Arizona, presenters recommended that cancer survivors be included in community education programs on cancer. Where feasible, presenters also noted that support groups for cancer patients and their families should be established (4).

To evaluate the effectiveness of these kinds of programs, a panel was invited to the Second National Conference on Cancer, which was held in Rapid City, South Dakota in September, 1992. This panel included Dr. Judith Salmon Kaur, two cancer survivors (N.J.S. and M.L.) and the widow (L.H.E.) of a cancer patient. The presentations given by these panelists brought a human dimension to the cancer statistics presented by other presenters.

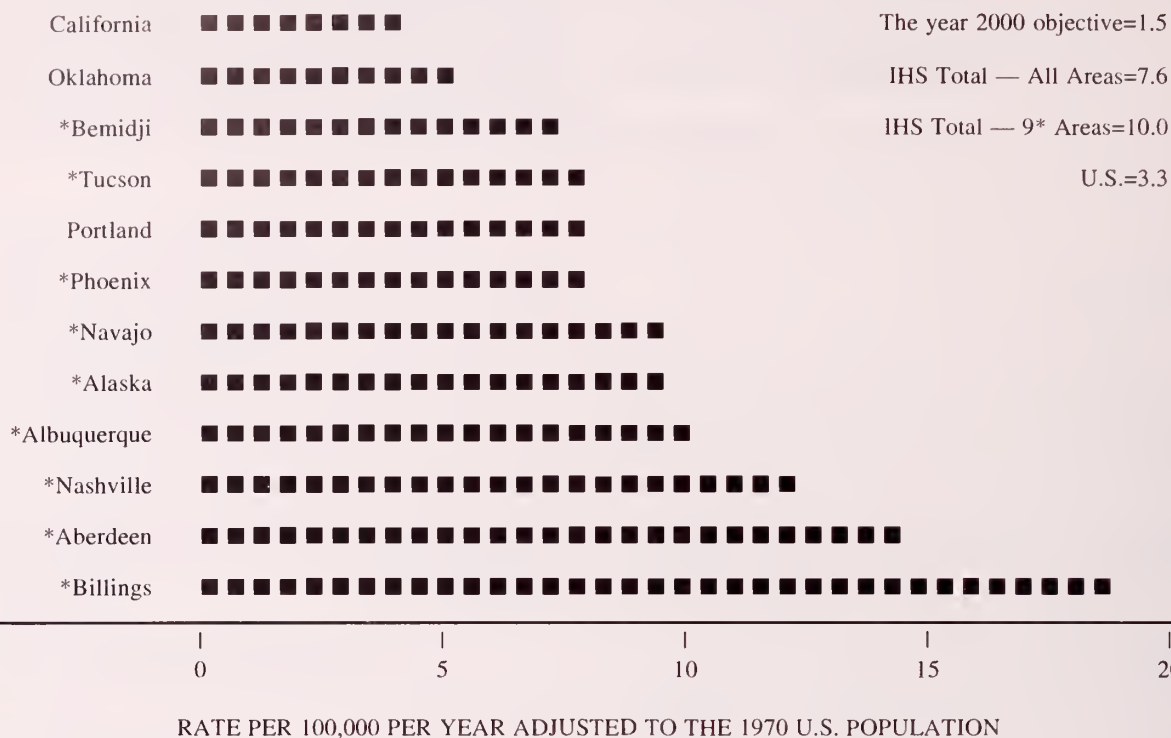
The first patient to share her story was N.J.S. from New Town, North Dakota. Her presentation emphasized the importance of screening and early detection. She spoke of her initial panic at learning she had breast cancer. Her surgeon involved her in the decision for lumpectomy and radiation. Her family supported her decision also, and now four years later she is disease free and a vocal advocate for women regarding mammography.

Dr. Bernie Siegel has written about the importance of patients taking control as part of the healing process rather than being a "victim" (5). N.J.S. had never known anyone on her reservation to survive cancer. As she informed the audience:



Figure 1

# Age-Adjusted Cervical Cancer Mortality Rates, by IHS Area, 1984-1988



\*Excluding California Oklahoma, and Portland Areas

For females, from 1984 to 1988, the age-adjusted cervical cancer mortality rate was 7.6/1000 for the entire IHS service population. When the 3 IHS Areas with apparent problems in underreporting of Indian race on death certificates are excluded, the rate was 10.0/1000. All IHS Areas had cervical cancer mortality rates higher than the U.S. rate, and 6 Areas had rates significantly higher than the U.S. rate. The Billings Area had the highest rate, 5.6 times the U.S. rate.<sup>1</sup>

<sup>1</sup> Information copied from Chart 32 in: Valway S (ed.). *Cancer Mortality Among Native Americans in the United States: Regional Differences in Indian Health, 1984-1988 and Trends Over Time, 1968-1987*. Rockville, Maryland: Indian Health Service, 1992.

It was really devastating for my kids because they thought I was holding back on them because they heard that if you had cancer that you were dying. They said, "Mom, are you sure you're telling us the truth?" I said, "Yes I am." I said, "I'm cured."

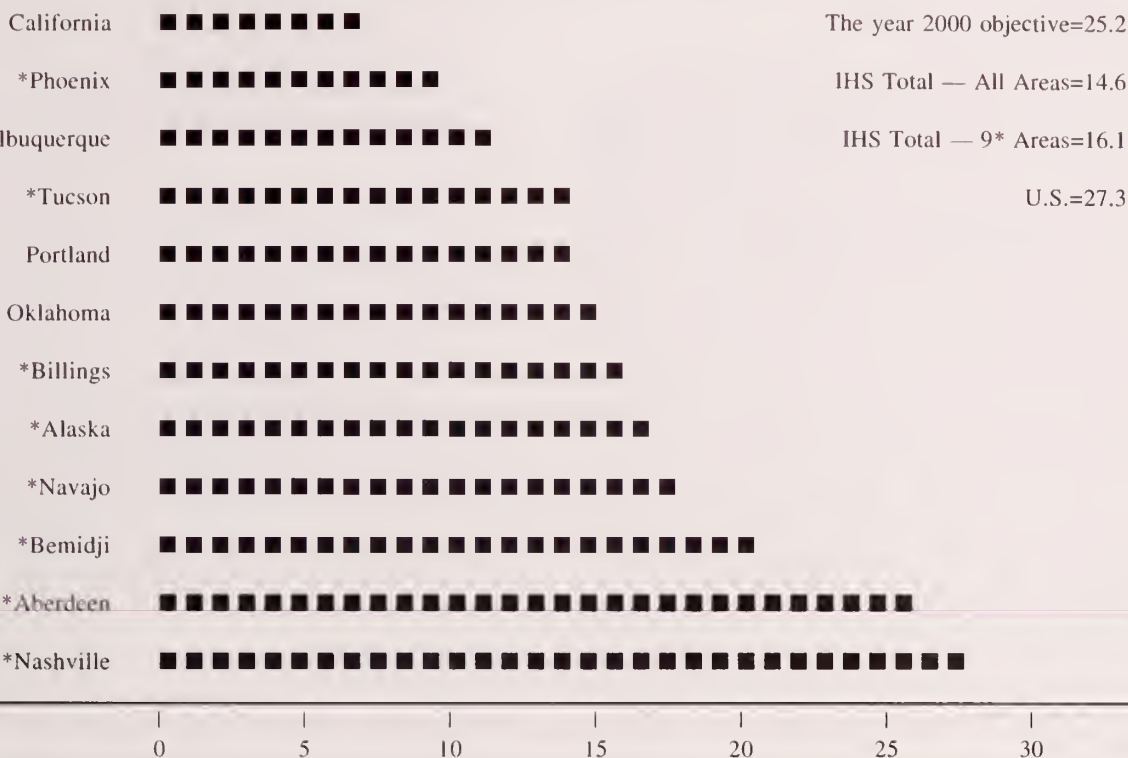
The older ones were having it rougher than the younger ones. I told them I'm too young to die. I love myself too much. I'm not going to die on you guys. Every six months I go in for check ups and they're all there waiting to see what the results are — it's like, well how are you? They make a joke of it now. Are you going to live six more months? They're dealing with it.

N.J.S.'s survival emphasizes the importance of screening and early detection. She is part of the "A su salud" program at Fort Berthold, where survivors serve as role models in their community to encourage healthy behaviors. This program was pioneered in Texas (6) and has been highly successful on the reservation. Her community now sees that breast cancer need not be a death sentence.

Our second speaker was M.L., a leukemia survivor now five years out from her bone marrow transplant. Her need for experimental treatment made her face the taboos within her pueblo and enlighten her people about cancer. Many people shunned her initially because of their fear of "contamination." She spoke eloquently about overcoming that fear by starting a support group for cancer patients and their families:

Figure 2

### Age-Adjusted Breast Cancer Mortality Rates, by IHS Area, Females, 1984-1988



RATE PER 100,000 PER YEAR ADJUSTED TO THE 1970 U.S. POPULATION

\*Excluding California Oklahoma, and Portland Areas

For females, from 1984 to 1988, the age-adjusted breast cancer mortality was 14.6/100,000 for the entire IHS service population. When the 3 IHS Areas with apparent problems in underreporting of Indian race on death certificates are excluded, the rate was 16.1/100,000.

All IHS Areas had breast cancer mortality rates for females that were lower than the U.S. rate, and 9 Areas had significantly lower rates. The California Area rate was the lowest, less than 1/4 the U.S. rate.<sup>2</sup>

<sup>2</sup> Information copied from Chart 31 in: Valway S (ed.). *Cancer Mortality Among Native Americans in the United States: Regional Differences in Indian Health, 1984-1988 and Trends Over Time, 1968-1987*. Rockville, Maryland: Indian Health Service, 1992.

They'd say don't touch her; she might be contagious for a long time. For a long time people would do that to me. Later on, I said I can't deal with this anymore. I told my family that when I get well, I'm going to start something with my life. My brother said, "Whoa! What are you talking about?" I said, "I'm going to start up a support group because everyone in the village feels that once you have cancer that you're going to die. At least that's how it was with my fate. They thought I was going to die,

but I didn't. I came back." My brother said, "Well, we'll support you in whatever you want to do." Then I went to the Pueblo government and told them what I wanted to do. They looked at me and said, "No." They said, "What are you trying to do? Kill everybody in the village?" I said, "No, I'm trying to organize something that will help the whole tribe." They said, "No, you can't do it. We don't allow people to do that, especially if they are patients with cancer. We can't allow you to do that." I went home in



tears, of course. . . . The following year, the first time I went back again there was a new governor, and he was a relation to me. He at least sat and listened to what I was saying. He said, "Okay."

Cella and Yellan (7) have noted the difficulty of launching support groups. Typically they are most utilized by white middle to upper class women as men often do not feel comfortable until a tangible product is offered (7). Nonetheless, M.L. overcame all these barriers in her community, and men and women alike attend the group. She had the courage to overcome time-honored perceptions of disease to become a survivor. Siegel has emphasized the "exceptional patient" who acts not only out of self-interest, but also from the interest of others. They give of themselves and make the world a better place than they found it (5). M.L. is certainly a perfect example of that model!

L.H.E., our final panelist, also participates in an ongoing support group off her reservation. Despite her grief over several losses of family members, she is able to share her spirituality and culture with both Native and non-Native group members:

I was brought up the traditional way. I just related to what Cecelia spoke about yesterday. My grandparents lived with us, and everything was prayer with them. My Grandma would pray even washing dishes. I'd say, "Grandma, who are you talking to?" She'd say, "I'm talking to the Great Spirit. He will provide at the Garden." Everything was prayer. So, this is what saved me.

She offered up a prayer in her native tongue for all conference attendees and emphasized the spiritual support needed to care for cancer patients and their grieving families.

The major accomplishments of this panel included the recognition of the need for tribal-based interventions, proof that support groups can be effective in a tribal setting, the potential usefulness that cross-cultural support groups offer, and the continued need to bring a human dimension to cancer statistics. Finally, the positive visible testimony of cancer survivors is a powerful tool for training tribal health officers and allied health personnel.

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# SUMMATION OF CONFERENCE

**James W. Hampton, M.D.**

The opening ceremonies included a welcome by Dr. Jennie Joe, a Navajo scientist, and Dr. Tom Welty, and epidemiologist, and a prayer by Mr. Sidney Keith, a Cheyenne River Sioux medicine man, which set the tone of the conference, "Cancer in Indian Country."

At the turn of the century cancer, was a "rare" disease for American Indian and Alaska Native people. Today, cancer has become an increasingly prominent cause of death for both sexes over age 30 and ranks second as a cause of death for those who are over age 45.

We need more accurate data on cancer incidence, mortality, and survival in Native Americans. Lung cancer is a leading cause of death in Plains Indian tribes and Alaska Native peoples, and matches the pattern of cigarette smoking — few Navajos smoke, few Navajos get lung cancer; many Lakotas smoke, and many Lakotas get lung cancer.

Cancer to American Indians means death, or "going to the other side." Prevention of cancer would mean keeping the "mind, heart and spirit clean." Dr. Harold Freeman in his keynote address, "Year 2000: Reducing Cancer in Special Populations" noted that "Native Americans should be empowered through their own culture to seek their assistance in cancer control practices." Information culturally targeted to the youth on smoke cessation and stopping the use of smokeless tobacco could reduce the cancers related to these practices.

American Indians are frequently obese, which is an indication of poor nutrition. About 28% of American Indian/Alaska Natives live in poverty, and poverty plays

a significant role in obesity. Many Indian people subsist on welfare commodities that are notoriously high in fat and low in fiber.

For the 61% of American Indian/Alaskan Native elders living in poverty, "navigators" could be designated to guide them through the maze of the health care system to assure that they receive "state of the art" cancer clinical management. As Dr. Freeman said, "poverty, starvation, and diseases of the Third World" exist in population "pockets" in our own country. The intelligence, technology, and financial support of our federal resources should be applied to our own people to improve the health care expectations of those "Third-World" Americans.

The barrier to the modern technology of cancer care is due in part to the culture of poverty and to the traditional American Indian lack of assertiveness. Cancer prevention in the traditional Indian culture can only be accomplished by empowerment of the people. Cancer survivors in this special population are being encouraged to speak up and tell their stories to their people in order to overcome the taboos about discussing cancer.

Unique cancers to this special population, such as carcinoma of the gallbladder and biliary tract, should be targeted for research. The survival of the almost 2 million indigenous people on the North American Continent after 500 years of decimation by European diseases is now threatened by cancer.

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# **PRAVACHOL® (Pravastatin Sodium Tablets)**

## **CONTRAINDICATIONS**

Hypersensitivity to any component of this medication.

Active liver disease or unexplained, persistent elevations in liver function tests (see WARNINGS).

**Pregnancy and lactation:** Atherosclerosis is a chronic process and discontinuation of lipid-lowering drugs during pregnancy should have little impact on the outcome of long-term therapy of primary hypercholesterolemia. Cholesterol and other products of cholesterol biosynthesis are essential components for fetal development (including synthesis of steroids and cell membranes). Since HMG-CoA reductase inhibitors decrease cholesterol synthesis and possibly the synthesis of other biologically active substances derived from cholesterol, they may cause fetal harm when administered to pregnant women. Therefore, HMG-CoA reductase inhibitors are contraindicated during pregnancy and in nursing mothers. **Pravastatin should be administered to women of childbearing age only when such patients are highly unlikely to conceive and have been informed of the potential hazards.** If the patient becomes pregnant while taking this class of drug, therapy should be discontinued and the patient apprised of the potential hazard to the fetus.

## **WARNINGS**

**Liver Enzymes:** HMG-CoA reductase inhibitors, like some other lipid-lowering therapies, have been associated with biochemical abnormalities of liver function. Increases of serum transaminase (ALT, AST) values to more than 3 times the upper limit of normal occurring on 2 or more (not necessarily sequential) occasions have been reported in 1.3% of patients treated with pravastatin in the U.S. over an average period of 18 months. These abnormalities were not associated with cholestasis and did not appear to be related to treatment duration. In those patients in whom these abnormalities were believed to be related to pravastatin and who were discontinued from therapy, the transaminase levels usually fell slowly to pretreatment levels. These biochemical findings are usually asymptomatic although worldwide experience indicates that anorexia, weakness, and/or abdominal pain may also be present in rare patients.

As with other lipid-lowering agents, liver function tests should be performed during therapy with pravastatin. Serum aminotransferases, including ALT (SGPT), should be monitored before treatment begins, every six weeks for the first three months, every eight weeks during the remainder of the first year, and periodically thereafter (e.g., at about six-month intervals). Special attention should be given to patients who develop increased transaminase levels. Liver function tests should be repeated to confirm an elevation and subsequently monitored at more frequent intervals. If increases in AST and ALT equal or exceed three times the upper limit of normal and persist, then therapy should be discontinued. Persistence of significant aminotransferase elevations following discontinuation of therapy may warrant consideration of liver biopsy.

Active liver disease or unexplained transaminase elevations are contraindications to the use of pravastatin (see CONTRAINDICATIONS). Caution should be exercised when pravastatin is administered to patients with a history of liver disease or heavy alcohol ingestion (see CLINICAL PHARMACOLOGY: Pharmacokinetics/Metabolism). Such patients should be closely monitored, started at the lower end of the recommended dosing range, and titrated to the desired therapeutic effect.

**Skeletal Muscle:** Rhabdomyolysis with renal dysfunction secondary to myoglobinuria has been reported with pravastatin and other drugs in this class. Uncomplicated myalgia has also been reported in pravastatin-treated patients (see ADVERSE REACTIONS). Myopathy, defined as muscle aching or muscle weakness in conjunction with increases in creatine phosphokinase (CPK) values to greater than 10 times the upper limit of normal was reported to be possibly due to pravastatin in only one patient in clinical trials (<0.1%). Myopathy should be considered in any patient with diffuse myalgias, muscle tenderness or weakness, and/or marked elevation of CPK. Patients should be advised to report promptly unexplained muscle pain, tenderness or weakness, particularly if accompanied by malaise or fever. **Pravastatin therapy should be discontinued if markedly elevated CPK levels occur or myopathy is diagnosed or suspected. Pravastatin therapy should also be temporarily withheld in any patient experiencing an acute or serious condition predisposing to the development of renal failure secondary to rhabdomyolysis, e.g., sepsis; hypotension; major surgery; trauma; severe metabolic, endocrine, or electrolyte disorders; or uncontrolled epilepsy.**

The risk of myopathy during treatment with lovastatin is increased if therapy with either cyclosporine, gemfibrozil, erythromycin, or niacin is administered concurrently. There is no experience with the use of pravastatin together with cyclosporine. Myopathy has not been observed in clinical trials involving small numbers of patients who were treated with pravastatin together with niacin. One trial of limited size involving combined therapy with pravastatin and gemfibrozil showed a trend toward more frequent CPK elevations and patient withdrawals due to musculoskeletal symptoms in the group receiving combined treatment as compared with the groups receiving placebo, gemfibrozil, or pravastatin monotherapy. Myopathy was not reported in this trial (see PRECAUTIONS: Drug Interactions). One patient developed myopathy when clofibrate was added to a previously well tolerated regimen of pravastatin; the myopathy resolved when clofibrate therapy was stopped and pravastatin treatment continued. **The use of fibrates alone may occasionally be associated with myopathy. The combined use of pravastatin and fibrates should generally be avoided.**

## **PRECAUTIONS**

**General:** Pravastatin may elevate creatine phosphokinase and transaminase levels (see ADVERSE REACTIONS). This should be considered in the differential diagnosis of chest pain in a patient on therapy with pravastatin.

**Homozygous Familial Hypercholesterolemia:** Pravastatin has not been evaluated in patients with rare homozygous familial hypercholesterolemia. In this group of patients, it has been reported that HMG-CoA reductase inhibitors are less effective because the patients lack functional LDL receptors.

**Renal Insufficiency:** A single 20 mg oral dose of pravastatin was administered to 24 patients with varying degrees of renal impairment (as determined by creatinine clearance). No effect was observed on the pharmacokinetics of pravastatin or its 3 $\alpha$ -hydroxy isomeric metabolite (SQ 31,906). A small increase was seen in mean AUC values and half-life (1/2) for the inactive enzymatic ring hydroxylation metabolite (SQ 31,945). Given this small sample size, the dosage administered, and the degree of individual variability, patients with renal impairment who are receiving pravastatin should be closely monitored.

**Information for Patients:** Patients should be advised to report promptly unexplained muscle pain, tenderness or weakness, particularly if accompanied by malaise or fever.

**Drug Interactions:** Immunosuppressive Drugs, Gemfibrozil, Niacin (Nicotinic Acid), Erythromycin. See WARNINGS: Skeletal Muscle.

**Antipyrine:** Clearance by the cytochrome P450 system was unaltered by concomitant administration of pravastatin. Since pravastatin does not appear to induce hepatic drug-metabolizing enzymes, it is not expected that any significant interaction of pravastatin with other drugs (e.g., phenytoin, quinidine) metabolized by the cytochrome P450 system will occur.

**Cholestyramine/Colestipol:** Concomitant administration resulted in an approximately 40 to 50% decrease in the mean AUC of pravastatin. However, when pravastatin was administered 1 hour before or 4 hours after cholestyramine or 1 hour before colestipol and a standard meal, there was no clinically significant decrease in bioavailability or therapeutic effect. (See DOSAGE AND ADMINISTRATION: Concomitant Therapy.)

**Warfarin:** In a study involving 10 healthy male subjects given pravastatin and warfarin concomitantly for 6 days, bioavailability parameters at steady state for pravastatin (parent compound) were not altered. Pravastatin did not alter the plasma protein-binding of warfarin. Concomitant dosing did increase the AUC and C<sub>max</sub> of warfarin but did not produce any changes in its anticoagulant action (i.e., no increase was seen in mean prothrombin time after 6 days of concomitant therapy). However, bleeding and extreme prolongation of prothrombin time has been reported with another drug in this class. Patients receiving warfarin-type anticoagulants should have their prothrombin times closely monitored when pravastatin is initiated or the dosage of pravastatin is changed.

**Cimetidine:** The AUC<sub>0-12hr</sub> for pravastatin when given with cimetidine was not significantly different from the AUC for pravastatin when given alone. A significant difference was observed between the AUC's for pravastatin when given with cimetidine compared to when administered with antacid.

**Digoxin:** In a crossover trial involving 18 healthy male subjects given pravastatin and digoxin concurrently for 9 days, the bioavailability parameters of digoxin were not affected. The AUC of pravastatin tended to increase, but the overall bioavailability of pravastatin plus its metabolites (SQ 31,906 and SQ 31,945 was not altered).

**Gemfibrozil:** In a crossover study in 20 healthy male volunteers given concomitant single doses of pravastatin and gemfibrozil, there was a significant decrease in urinary excretion and protein binding of pravastatin. In addition, there was a significant increase in AUC, C<sub>max</sub>, and T<sub>max</sub> for the pravastatin metabolite SQ 31,906. Combination therapy with pravastatin and gemfibrozil is generally not recommended.

In interaction studies with aspirin, antacids 1 hour prior to PRAVACHOL (pravastatin sodium), cimetidine, nicotinic acid, or probucol, no statistically significant differences in bioavailability were seen when PRAVACHOL was administered.

**Other Drugs:** During clinical trials, no noticeable drug interactions were reported when PRAVACHOL was added to diuretics, antihypertensives, digitalis, converting-enzyme inhibitors, calcium channel blockers, beta-blockers, or nitroglycerin.

**Endocrine Function:** HMG-CoA reductase inhibitors interfere with cholesterol synthesis and lower circulating cholesterol levels and, as such, might theoretically blunt adrenal or gonadal steroid hormone production. Results of clinical trials with pravastatin in males and post-menopausal females were inconsistent with regard to possible effects of the drug on basal steroid hormone levels. In a study of 21 males, the mean testosterone response to human chorionic gonadotropin was significantly reduced (p<0.004) after 16 weeks of treatment with 40 mg of pravastatin. However, the percentage of patients showing a  $\geq$ 50% rise in plasma testosterone after human chorionic gonadotropin stimulation did not change significantly after therapy in these patients. The effects of HMG-CoA reductase inhibitors on spermatogenesis and fertility have not been studied in adequate numbers of patients. The effects, if any, of pravastatin on the pituitary-gonadal axis in pre-menopausal females are unknown. Patients treated with pravastatin who display clinical evidence of endocrine dysfunction should be evaluated appropriately. Caution should also be exercised if an HMG-CoA reductase inhibitor or other agent used to lower cholesterol levels is administered to patients also receiving other drugs (e.g., ketoconazole, spironolactone, cimetidine) that may diminish the levels or activity of steroid hormones.

**CNS Toxicity:** CNS vascular lesions, characterized by perivascular hemorrhage and edema and mononuclear cell

infiltration of perivascular spaces, were seen in dogs treated with pravastatin at a dose of 25 mg/kg/day, a dose that produced a plasma drug level about 50 times higher than the mean drug level in humans taking 40 mg/day. Similar CNS vascular lesions have been observed with several other drugs in this class.

A chemically similar drug in this class produced optic nerve degeneration (Wallerian degeneration of retinoganglionic fibers) in clinically normal dogs in a dose-dependent fashion starting at 60 mg/kg/day, a dose that produced mean plasma drug levels about 30 times higher than the mean drug level in humans taking the highest recommended dose (as measured by total enzyme inhibitory activity). This same drug also produced vestibulocochlear (Wallerian-like) degeneration and retinal ganglion cell chromatolysis in dogs treated for 14 weeks at 180 mg/kg/day, a dose which resulted in a mean plasma drug level similar to that seen with the 60 mg/kg dose.

**Carcinogenesis, Mutagenesis, Impairment of Fertility:** In a 2-year study in rats fed pravastatin at doses of 10, 30, or 100 mg/kg body weight, there was an increased incidence of hepatocellular carcinomas in males at the highest dose (p<0.01). Although rats were given up to 125 times the human dose (HD) on a mg/kg body weight basis, their serum drug levels were only 6 to 10 times higher than those measured in humans given 40 mg pravastatin as measured by AUC.

The oral administration of 10, 30, or 100 mg/kg (producing plasma drug levels approximately 0.5 to 5.0 times human drug levels at 40 mg) of pravastatin to mice for 22 months resulted in a statistically significant increase in the incidence of malignant lymphomas in treated females when all treatment groups were pooled and compared to controls (p<0.05). The incidence was not dose-related and male mice were not affected.

A chemically similar drug in this class was administered to mice for 72 weeks at 25, 100, and 400 mg/kg body weight, which resulted in mean serum drug levels approximately 3, 15, and 33 times higher than the mean human serum drug concentration (as total inhibitory activity) after a 40 mg oral dose. Liver carcinomas were significantly increased in high-dose females and mid- and high-dose males, with a maximum incidence of 90 percent in males. The incidence of adenomas of the liver was significantly increased in mid- and high-dose females. Drug treatment also significantly increased the incidence of lung adenomas in mid- and high-dose males and females. Adenomas of the eye Harderian gland (a gland of the eye of rodents) were significantly higher in high-dose mice than in controls.

No evidence of mutagenicity was observed *in vitro*, with or without rat liver metabolic activation, in the following studies: microbial mutagen tests, using mutant strains of *Salmonella typhimurium* or *Escherichia coli*, a forward mutation assay in L5178Y TK +/– mouse lymphoma cells, a chromosomal aberration test in hamster cells, and a gene conversion assay using *Saccharomyces cerevisiae*. In addition, there was no evidence of mutagenicity in either a dominant lethal test in mice or a micronucleus test in mice.

In a study in rats, with daily doses up to 500 mg/kg, pravastatin did not produce any adverse effects on fertility or general reproductive performance. However, in a study with another HMG-CoA reductase inhibitor, there was decreased fertility in male rats treated for 34 weeks at 25 mg/kg body weight, although this effect was not observed in a subsequent fertility study when this same dose was administered for 11 weeks (the entire cycle of spermatogenesis, including epididymal maturation). In rats treated with this same reductase inhibitor at 180 mg/kg/day, seminiferous tubule degeneration (necrosis and loss of spermatogenic epithelium) was observed. Although not seen with pravastatin, two similar drugs in this class caused drug-related testicular atrophy, decreased spermatogenesis, spermatocytic degeneration, and giant cell formation in dogs. The clinical significance of these findings is unclear.

**Pregnancy: Pregnancy Category X:** See CONTRAINDICATIONS.

Safety in pregnant women has not been established. Pravastatin was not teratogenic in rats at doses up to 1000 mg/kg daily or in rabbits at doses of up to 50 mg/kg daily. These doses resulted in 20x (rabbit) or 240x (rat) the human exposure based on surface area (mg/m<sup>2</sup>). However, in studies with another HMG-CoA reductase inhibitor, skeletal malformations were observed in rats and mice. PRAVACHOL (pravastatin sodium) should be administered to women of child-bearing potential only when such patients are highly unlikely to conceive and have been informed of the potential hazards. If the woman becomes pregnant while taking PRAVACHOL, it should be discontinued and the patient advised again as to the potential hazards to the fetus.

**Nursing Mothers:** A small amount of pravastatin is excreted in human breast milk. Because of the potential for serious adverse reactions in nursing infants, women taking PRAVACHOL should not nurse (see CONTRAINDICATIONS).

**Pediatric Use:** Safety and effectiveness in individuals less than 18 years old have not been established. Hence, treatment in patients less than 18 years old is not recommended at this time. (See also PRECAUTIONS: General.)

## **ADVERSE REACTIONS**

Pravastatin is generally well tolerated, adverse reactions have usually been mild and transient. In 4-month long placebo-controlled trials, 1.7% of pravastatin-treated patients and 1.2% of placebo-treated patients were discontinued from treatment because of adverse experiences attributed to study drug therapy. This difference was not statistically significant. In long-term studies, the most common reasons for discontinuation were asymptomatic serum transaminase increases and mild, non-specific gastrointestinal complaints. During clinical trials the overall incidence of adverse events in the elderly was not different from the incidence observed in younger patients.

**Adverse Clinical Events:** All adverse clinical events (regardless of attribution) reported in more than 2% of pravastatin-treated patients in the placebo-controlled trials are identified in the table below, also shown are the percentages of patients in whom these medical events were believed to be related or possibly related to the drug.

Body System/Event	All Events %		Events Attributed to Study Drug %	
	Pravastatin (N=900)	Placebo (N=411)	Pravastatin (N=900)	Placebo (N=411)
Cardiovascular				
Cardiac Chest Pain	4.0	3.4	0.1	0.0
Dermatologic				
Rash	4.0*	1.1	1.3	0.9
Gastrointestinal				
Nausea/Vomiting	7.3	7.1	2.9	3.4
Diarrhea	6.2	5.6	2.0	1.9
Abdominal Pain	5.4	6.9	2.0	3.9
Constipation	4.0	7.1	2.4	3.1
Flatulence	3.3	3.6	2.7	3.4
Heartburn	2.9	1.9	2.0	0.7
General				
Fatigue	3.8	3.4	1.9	1.0
Chest Pain	3.7	1.9	0.3	0.2
Influenza	2.4*	0.7	0.0	0.0
Musculoskeletal				
Localized Pain	10.0	9.0	1.6	1.5
Myalgia	2.7	1.0	0.6	0.0
Nervous System				
Headache	6.2	3.9	1.7*	0.2
Dizziness	3.3	3.2	1.0	0.5
Renal/Genitourinary				
Urinary Abnormality	2.4	2.9	0.7	1.2
Respiratory				
Common Cold	7.0	6.3	0.0	0.0
Rhinitis	4.0	4.1	0.1	0.0
Cough	2.6	1.7	0.1	0.0

\*Statistically significantly different from placebo.

The following effects have been reported with drugs in this class:

Skeletal myopathy, rhabdomyolysis

Neurological: dysfunction of certain cranial nerves (including alteration of taste, impairment of extra-ocular movement, facial palsy); tremor, vertigo, memory loss, paresthesia, peripheral neuropathy, peripheral nerve palsy.

Hypersensitivity Reactions: An apparent hypersensitivity syndrome has been reported rarely which has included one or more of the following features: anaphylaxis, angioedema, lupus erythematosus-like syndrome, polymyalgia rheumatica, vasculitis, purpura, thrombocytopenia, leukopenia, hemolytic anemia, positive ANA, ESR increase, arthritis, arthralgia, urticaria, asthenia, photosensitivity, fever, chills, flushing, malaise, dyspnea, toxic epidermal necrolysis, erythema multiforme, including Stevens-Johnson syndrome.

Gastrointestinal: pancreatitis, hepatitis, including chronic active hepatitis, cholestatic jaundice, fatty change in liver, and, rarely, cirrhosis, fulminant hepatic necrosis, and hepatoma; anorexia, vomiting.

Reproductive: gynecomastia, loss of libido, erectile dysfunction.

Eye: progression of cataracts (lens opacities), ophthalmoplegia

**Laboratory Test Abnormalities:** Increases in serum transaminase (ALT, AST) values and CPK have been observed (see WARNINGS).

Transient, asymptomatic eosinophilia has been reported. Eosinophil counts usually returned to normal despite continued therapy. Anemia, thrombocytopenia, and leukopenia have been reported with other HMG-CoA reductase inhibitors.

**Concomitant Therapy:** Pravastatin has been administered concurrently with cholestyramine, colestipol, nicotinic acid, probucol and gemfibrozil. Preliminary data suggest that the addition of either probucol or gemfibrozil to therapy with lovastatin or pravastatin is not associated with greater reduction in LDL-cholesterol than that achieved with lovastatin or pravastatin alone. No adverse reactions unique to the combination or in addition to those previously reported for each drug alone have been reported. Myopathy and rhabdomyolysis (with or without acute renal failure) have been reported when another HMG-CoA reductase inhibitor was used in combination with immunosuppressive drugs, gemfibrozil, erythromycin, or lipid-lowering doses of nicotinic acid. Concomitant therapy with HMG-CoA reductase inhibitors and these agents is generally not recommended. (See WARNINGS: Skeletal Muscle and PRECAUTIONS: Drug Interactions.)


## **OVERDOSAGE**

There have been no reports of overdoses with pravastatin.

Should an accidental overdose occur, treat symptomatically and institute supportive measures as required.



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**CONTRAINDICATIONS, WARNINGS, PRECAUTIONS, and ADVERSE REACTIONS** in the brief prescribing information on the adjacent page.

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# ALASKA MEDICINE

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***In this issue:* Cancer in the Alaska Native Population: Eskimo, Aleut and Indian  
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## CANCER IN ALASKA NATIVES

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# **CANCER IN THE ALASKA NATIVE POPULATION: ESKIMO, ALEUT, AND INDIAN INCIDENCE AND TRENDS 1969 - 1988**

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In 1989, a new collaboration was established with the New Mexico Tumor Registry, University of New Mexico Medical Center, Albuquerque, New Mexico. Charles Key, MD, Anna Marie Davidson, Ron Darling and numerous Certified Tumor Registrars at NMTR provided technical assistance, training and record abstracting. They also made available the personal computer-based registry software developed at the University of New Mexico, willingly adapted the program to fit Alaskan needs, and were readily available for consultation when needed.

The Tumor Registry is located in the Department of Medicine, Alaska Native Medical Center. We wish to thank the secretarial staff of the department and Dennis Beckworth, MD, oncologist. The personal efforts of Bonnie Smith, Claudette Amadon, Annette Harpster and Helen Peters deserve special mention.

*The opinions expressed in this report are those of the authors and do not necessarily reflect the views of the Indian Health Service.*

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## FOREWORD

Cancer is now a major problem among Alaska Native people. It is *the leading* cause of death among women, and third among men. It is the third most frequent cause of hospital days for illness among Indian Health Service beneficiaries served by the Alaska Area Native Health Service. Alaska Native Elders report that cancer was not a common form of disease among the population in the past. Unfortunately nearly every Native family and community has now experienced first hand the suffering and death from this disease.

The information presented in this report are some of the data compiled by the Alaska Native Cancer Surveillance Project, also referred to as the Alaska Native Tumor Registry. Funding for this project has come from the National Cancer Institute, originally from the Division of Cancer Etiology, and currently from the Division of Cancer Prevention and Control.

The cancer information included in this report is "incidence" data, that is, *new* cases of cancers, in contrast to "mortality" or deaths from cancer. Incidence data are now retained at the Alaska Native Medical Center, Anchorage. Death data are compiled by the State of Alaska, Office of Vital Statistics, Juneau and submitted to the National Center for Health Statistics.

The cancer incidence data for the Alaska Native people in this report cover the twenty year period, 1969-

88. To the best of our knowledge, the data are complete statewide for all Alaska Natives/American Indians residents of Alaska at the time of diagnosis of cancer. Data for the years 1989 to 1993 are currently being compiled. Alaska hospitals participated in the Washington/Alaska Automated Tumor Registry in the late 1960s and the early 1970s. The State of Alaska maintained an Alaska Tumor Registry for one year, 1974. Those data supported the clinical impression that Alaska Natives experienced unusual cancer patterns. Therefore, efforts were made to continue cancer surveillance of Alaska Native people. As a result, complete statewide incidence data are available from 1969 to the present, but only for Alaska Natives.

The information presented in this report is just a portion of that collected by the Alaska Native Cancer Surveillance Project. Data are formatted to make as much information available to the Alaska Native people, as well as to health providers, planners, administrators, etc. Additional data will be made available as soon as possible. Comments and questions on this information, and suggestions regarding future reports should be directed to: Anne P. Lanier, MD, MPH, Alaska Area Native Health Service, Office of Community Health Services, Epidemiology and Cancer Control, 250 Gambell Street, Anchorage, Alaska 99501.

# PROJECT SUMMARY

## CANCER INCIDENCE AND TRENDS AMONG ALASKA NATIVES 1969-1988

### INTRODUCTION

Statewide cancer incidence rates for Alaska Natives (Eskimo, Aleut, Indian) were first reported in 1976 based on identification of newly diagnosed patients for the five year time period 1969-73 (1). The number of newly diagnosed cases for all cancers combined were approximately 15% lower in Alaska Native men and women than in the U.S. population for the same time period, after adjustments were made for the young age of the Alaska Native population.

Site-specific cancer patterns in Alaska Natives differed markedly from those of other U.S. populations including American Indians in the other 49 states. Alaska Natives were at significantly increased risk for cancers of the nasopharynx, salivary gland, liver, gallbladder, kidney and thyroid. Significant deficits were noted for cancers of the prostate, breast, uterus, and melanoma. Rates for leukemia and lymphoma were also low. Subsequent reports confirmed the unusual cancer patterns (2-5). The data also showed variation by ethnic group within the Alaska Native population (6). In general, rates for breast and prostate cancer were higher among the Indian than the Eskimo population. On the other hand, cancer rates of the nasopharynx, esophagus, liver and gallbladder were higher in Eskimos than Indians.

This summary includes information available on cancer incidence in Alaska Natives for the twenty year time period (1969-88). Cancers included in this report are those routinely identified and reported by tumor registries; that is, *invasive* cancers (exclusive of basal and squamous cell skin cancers). Cancers characterized by the pathologists as "in situ" or "intraepithelial neoplasia" are not included here.

Some tables and graphs show data only for 1984-88. These data best portray the current cancer patterns in the population.

### METHODS

For the twenty year period 1969-88 Alaska Native patients diagnosed with cancer while residents of the state of Alaska were identified statewide. Background information on the population and methods for cancer identification and registration have been previously described (1,6). In general, all public and private facilities for identification, diagnosis, and treatment of Alaska Native cancer patients were surveyed. This included

intensive review of: hospital inpatient and outpatient diagnostic indices, pathology reports, death certificates, and tumor registries when available. Data items were collected in accordance with National Cancer Institute's Statistics, Epidemiology and End Results (SEER) Program (7). Patients were considered Alaska Native if they had documentation of eligibility for medical care from the Indian Health Service (IHS). Classification of Eskimo, Indian, or Aleut is part of the IHS medical record and is based on self designation.

Population data are from the 1970, 1980 and 1990 U.S. Census. The Alaska Native population increased from 50,900 in 1970 to 85,698 in 1990. Alaska Native cancer rates were age-adjusted to the U.S. 1970 standard population. Rates were tested for significant differences (8). Trends over time were examined by calculating average annual rates for each of four five-year time periods (1969-73, 1974-78, 1979-83, and 1984-88), and tested for significant changes over time (9).

### RESULTS

The number of cases and rates by sex, site, and time period are shown in Tables 1 and 2. A total of 2,318 invasive cancers in Alaska Natives who were residents of Alaska at the time of diagnosis were identified in the twenty year period, 1969-88. Thirty-six percent (829) of all cancers were identified in the last five years. Of all the cases, 94% were confirmed by histology or cytology. The number of cases more than doubled from the first to the last of the four five-year time periods. The number of cases in men and women is nearly equal for the entire 20 year period, but there has been a shift from a male predominance prior to 1979 (M:F ratio = 1.1:1) to a female predominance in the most recent time period 1984-88 (M:F = 1:1.2).

### Age-specific rates

Data for the entire twenty year period were used to calculate average annual age-specific rates for both sexes for all cancers combined and for each site (Tables 3 and 4). For most cancer sites, rates begin to rise in the fifth decade of life and continue to increase with age. Exceptions to this pattern are seen for cancer of the cervix and uterus where rates level off after the sixth decade. Some cancers which occur under age 30 and have rates that are quite high include: stomach, liver, breast, cervix, ovary, brain, thyroid and leukemia.



## Cancer trends

Trends were examined based on the average annual age-adjusted rates for all cancers and each cancer site by sex and each of four five-year time periods (Tables 1 and 2). From the first to the last time period, rates for all cancers combined increased 10% in men from 303 to 333 per 100,000, while rates in women increased 34% (310 to 415 per 100,000). Both these increases were statistically significant. The most dramatic increase was noted for lung cancer - two-fold in men, and five-fold in women. Significant increases were also noted in rates in women for cancer of the colon/rectum and breast. Kidney cancer increased in both men and women; the increase was significant if data for both men and women were combined. Cancers of the prostate and cervix appeared to increase over the first three time periods, 1969-83, then decrease in the last five years.

## Rates by ethnic group

Age-adjusted rates were calculated for each ethnic group (Eskimo, Aleut, Indian) for all cancers combined and for separate cancer sites using data for the entire twenty year period (Tables 5 and 6). Rates for each sex and both sexes combined for each ethnic group were tested for significant differences. No differences were found for cancers of the lung and cervix. Cancer rates of the colon/rectum were higher in Aleuts and Eskimos than Indians. Rates for cancer of the prostate were higher in Indians than both Eskimos and Aleuts.

## Rank order of cancers

Data for the most recent five year period, 1984-88, were used to determine rank order. Lung cancer is the most frequently occurring cancer in men and women combined and comprises 21% of all new cancers diagnosed. For men and women combined, cancer of the lung is followed by colon/rectum (18%), breast (11%), kidney (5%), stomach (5%), prostate (4%), cervix (3%), and pancreas (3%) cancer. In men the most frequently diagnosed cancers are lung (26%), colon/rectum (18%), prostate (8%), stomach (7%), oral cavity (6%), and kidney (5%). In women the top ranking cancers are breast (20%), colon/rectum (19%), lung (17%), cervix (6%), and kidney (5%).

## Comparison of cancer in all Alaska Natives with U.S.

Table 7 shows the numbers of cancers and rates for Alaska Natives for the most recent five year period compared with rates for U.S. Whites. These are shown as a rate ratio; a ratio higher than one means Natives have an excess, lower than one indicates a relative deficit compared to the U.S.. Based on cancer incidence data for this time period, the rate ratios of age-adjusted Alaska Native rates compared to U.S. Whites are 1.0 for both sexes combined, 0.8 for men and 1.2 for women.

Both the latter differences are significantly different from one. When each of the sites are analyzed separately, rate ratios are significantly high in Natives for one or both sexes for cancers of the: oral cavity, salivary gland, nasopharynx, stomach, colon/rectum, liver, gallbladder, lung, cervix, kidney and thyroid. On the other hand, ratios are low for larynx, melanoma of the skin, breast, uterus, prostate, bladder, eye, brain, lymphoma and leukemia.

## Comparison of cancer in ethnic groups with U.S.

In order to compare rates for each ethnic group with U.S. Whites, rates were also calculated using the most recent data (1984-88) for all cancers combined and the more frequently diagnosed sites (Table 8). In comparison with U.S. Whites, rate ratios were significantly low for prostate cancer in all three ethnic groups. Ratios were significantly low for breast cancer in Eskimos and also in Aleuts, but not Indians. Cervical cancer ratios were high in Indians and Eskimos, and significantly high in the Eskimos. Rate ratios for lung and colon/rectum cancer were high in women in all three ethnic groups.

## DISCUSSION

The data presented in this report support the following conclusions. One, the burden of cancer in the Alaska Native population now equals or exceeds that of U.S. Whites. The rate ratio of age-adjusted rates for Alaska Natives compared to U.S. Whites equals one. This is particularly noteworthy since little more than a generation ago, cancer was thought to be a rare disease in this population (10). Lung cancer, now the most frequently diagnosed cancer, was especially rare a generation ago (11). The ratios of cancer mortality (death) in Alaska Natives compared to U.S. Whites are higher than those for incidence (12) and are due to a relatively higher proportion of cancers with poor survival rates among the Alaska Native population. This may reflect late stage at diagnosis, or poorer survival even when adjusted for stage and treatment, as was found among American Indians in New Mexico (13). Analysis of stage at diagnosis and survival rates from cancer in the Alaska Native population are now in progress.

Two, the burden of cancer in Alaska Native women is disproportionately higher than in men. Age-adjusted rates in Alaska Native women exceed those in men (414 vs. 333 per 100,000), while the opposite is true in U.S. Whites (men - 433, women - 340 per 100,000). Ratios of rates for Alaska Natives and U.S. Whites are 1.2 in women and 0.8 in men. This burden of cancer in Alaska Native women is even more remarkable in view of the low rate for breast cancer in Alaska Native women compared to U.S. White women. Among all patients with cancer, there has been a shift over the twenty year

period from a male excess to a female excess of cancers. The ratio of males to females in the Native population in general is nearly equal.

Three, although incidence rates for all cancers combined in Alaska Natives now equal those of U.S. Whites, site-specific cancer incidence data differ markedly. Report data show that Alaska Natives continue to be at increased risk for cancers of the nasopharynx, salivary gland, esophagus, stomach, liver, gallbladder, cervix and kidney. Current data also indicate men and women are at high risk for cancer of the stomach. Women are also at high risk for cancers of the colon/rectum and lung. The Alaska Native population is at low risk for cancers of the larynx, breast, uterus, prostate, bladder, brain, leukemia, lymphoma and multiple myeloma.

Four, cancer patterns in Alaska Natives differ not only from those of U.S. Whites, but from other U.S. populations and Asian populations as well (13). Cancer patterns in this population also differ from American Indians (14). Cancer patterns in Alaska Natives not only differ from those of other populations but are rapidly changing. It is important to continue to collect accurate cancer data on the Alaska Native population, since incidence, trends, and survival cannot be predicted from national cancer databases.

Five, although the ratios of rates in Alaska Natives compared to U.S. Whites by cancer site differ markedly, there are similarities in rank order. Based on 1984-88 data, certain cancers now occur as frequently in the Alaska Native as in other U.S. populations. Among Alaska Natives, the most frequently diagnosed cancers are lung, colon/rectum, and breast in both sexes combined. These three cancers account for 50% of the total cancers. In Alaska Native men, the three most frequently diagnosed cancers — lung, colon/rectum, prostate account for 50% of the total male cancers, while the three highest ranking cancers in women — breast, colon/rectum, lung are responsible for 56% of the total. Thus, effective programs focused on prevention and decreased morbidity and mortality from these cancers will have the largest impact on the cancer burden.

Six, data for 1969-88 document increasing trends in cancer in this population. Increases in cancer are most marked in *women* and in *lung* cancer. Lung cancer in women increased five-fold in the twenty year period. Increases are also seen in kidney cancer (both sexes) and breast and colon/rectum (women). There is little evidence for declining rates in any cancer.

Finally, these data emphasize important points regarding cancer prevention and control in this population. Rates of cancer among Alaska Natives are increasing. The cancer which is increasing most rapidly, and responsible for much of the increase in total cancers, is lung cancer. Ninety percent of lung cancers

are caused by tobacco smoke and thus PREVENTABLE. Increasing lung cancer rates are consistent with increased availability and use of cigarettes following World War II, first in men and then in women. The prevalence of current smokers in the adult Native population is 50% in women and men (15,16). Use of spitting tobacco (snuff, chew, smokeless) is also high and begins at a very young age (16). Spitting tobacco is a particular problem. Not only does it cause cancer itself, but it also contributes to the cancer burden by causing early nicotine addiction and subsequent increased use of cigarettes. Since cigarette use is responsible for 90% of lung cancers, *and* a proportion of many other cancers (oral, esophagus, stomach, pancreas, larynx, cervix, bladder, leukemia), these cancers will continue to occur frequently until there is a significant reduction in tobacco use.

Another factor that can reduce cancer risk and improve survival with treatment is proper nutrition. A diet low in fat and high in fruits and vegetables is recommended to reduce cancer risk (17,18). This is achievable in the Alaska Native population using traditional subsistence foods, including fish and lean game, wild berries and plants. Limiting alcohol use will also reduce the risk for many cancers. Reduction of exposure to sexually transmitted diseases will reduce genital cancers.

Although not preventable at this time, several cancers can be detected early by screening and be cured. Regular self examination (of the breasts and testes); examinations by providers (breast, testes, rectum, prostate), and screening tests (Pap smear for cervix, mammogram of breast) can improve the stage at diagnosis, treatment effectiveness and survival. Persons with a family history of cancer should be particularly concerned about their risk factors and closely follow recommendations for cancer screening.

Since the first incidence survey, Alaska Natives have been known to be at increased risk for liver cancer. A successful primary and secondary cancer prevention program for liver cancer has been ongoing since 1985 (19). This includes hepatitis B vaccination of all serosusceptible individuals and newborns, and serum alpha-fetoprotein testing biannually of hepatitis B surface antigen carriers. This program has already resulted in improvement in five year survival from less than 25% to 75% for persons diagnosed with liver cancer.

Accurate monitoring of cancer incidence patterns in this population has documented that cancer patterns are unique and changing. These data have helped to identify areas for focus of interventions common in other populations (e.g. screening Pap tests and mammogram), as well as unique cancer control programs (e.g. liver cancer). Hopefully, accurate documentation of cancer patterns in this population will also help identify the causes of cancer, not only in this population, but in other populations as well.



**Table 1. Invasive Cancers in Alaska Natives 1969-1988  
Average Annual Incidence Rates (Age-Adjusted<sup>+</sup>) per 100,000  
by 5 Year Time Periods, Males**

Site	1969-73		1974-78		1979-83		1984-88	
	No.	Rate	No.	Rate	No.	Rate	No.	Rate
All Sites	207	302.9	251	299.7	308	324.2	372	332.7*
Oral Cavity & Pharynx	14	16.5	22	22.9	9	8.8	23	19.1
Salivary Gland	3	3.1	0	0.0	1	1.0	1	1.0
Nasopharynx	9	11.1	17	17.8	6	6.5	11	8.9
Gum & Other Oral Cavity	0	0.0	2	2.0	2	1.3	3	2.1
Digestive	77	112.7	88	109.2	111	116.9	132	118.9
Esophagus	6	11.3	5	6.8	7	7.7	7	7.0
Stomach	12	19.6	17	20.9	25	25.4	26	22.2
Colon & Rectum	36	60.4	45	57.5	46	51.1	66	61.9
Colon	22	37.4	29	36.0	36	40.7	47	44.4
Rectosigmoid Junction	3	5.6	6	7.6	3	2.1	5	4.4
Rectum	11	17.4	10	13.9	7	8.2	14	13.2
Liver	9	9.1	10	9.9	13	11.8	11	7.7
Gallbladder	2	4.3	2	3.1	5	5.8	1	1.0
Other Biliary	2	3.5	1	1.1	0	0.0	7	6.9
Pancreas	8	11.9	7	8.6	9	9.1	12	10.3
Respiratory	36	50.4	57	73.6	73	79.0	102	95.5*
Lung & Bronchus	34	47.9	55	71.8	66	72.7	97	91.4*
Larynx	0	0.0	1	0.9	5	4.8	1	0.8
Bones & Joints	4	5.5	1	0.9	0	0.0	5	3.1
Soft Tissue	4	6.4	4	3.3	0	0.0	2	1.7
Melanoma of Skin	0	0.0	1	0.9	1	0.5	0	0.0
Breast	0	0.0	0	0.0	1	1.1	0	0.0
Male Genital	23	34.5	23	27.9	41	44.3	41	36.3
Prostate	16	27.5	14	21.0	35	41.1	31	31.1
Testis	4	3.1	8	5.7	5	2.7	8	3.7
Urinary	15	21.4	16	19.3	14	16.3	27	25.4
Bladder	7	10.3	5	6.5	6	7.3	7	6.2
Kidney & Renal Pelvis	8	11.1	10	11.2	8	9.0	20	19.1
Eye & Orbit	0	0.0	0	0.0	3	2.0	0	0.0
Brain & Nervous System	3	1.8	5	4.4	3	1.8	4	2.2
Brain	3	1.8	5	4.4	3	1.8	4	2.2
Endocrine	0	0.0	5	5.1	5	5.4	4	2.8
Thyroid	0	0.0	4	4.0	3	3.4	2	1.5
Lymphomas	5	5.9	4	3.8	18	18.0	9	7.5
Hodgkin's	0	0.0	1	0.5	2	1.3	1	0.4
Non-Hodgkin's	5	5.9	3	3.3	16	16.6	8	7.1
Multiple Myeloma	4	5.7	3	4.4	4	4.6	2	2.0
Leukemia	7	8.4	5	3.9	5	3.2	8	4.7
Ill Defined & Unspecified	15	23.8	17	20.2	20	22.5	13	10.8

+Rates are age-adjusted to 1970 US standard population

\*Mantel-Haenszel chi-square for trend,  $p < 0.05$ .

**Table 2. Invasive Cancers in Alaska Natives 1969-1988**  
**Average Annual Incidence Rates (Age-Adjusted<sup>+</sup>) per 100,000**  
**by 5 Year Time Periods, Females**

Site	1969-73		1974-78		1979-83		1984-88	
	No.	Rate	No.	Rate	No.	Rate	No.	Rate
All Sites	187	309.8	235	329.3	301	344.2	453	414.5*
Oral Cavity & Pharynx	13	17.2	12	14.4	15	18.3	14	13.4
Salivary Gland	5	4.9	3	3.5	1	1.1	3	2.1
Nasopharynx	3	3.9	7	9.0	6	6.5	6	5.6
Gum & Other Oral Cavity	2	4.0	0	0.0	4	5.8	1	1.2
Digestive	67	136.7	67	114.0	90	119.4	136	137.6*
Esophagus	1	1.9	3	4.8	3	4.4	5	5.3
Stomach	6	10.7	4	5.3	6	7.6	14	12.0
Colon & Rectum	37	74.6	39	71.0	56	75.2	86	89.3*
Colon	25	51.2	32	58.7	43	56.6	70	72.3*
Rectosigmoid Junction	3	5.9	2	3.6	6	8.1	5	6.0
Rectum	9	17.5	5	8.7	7	10.5	11	11.0
Liver	1	2.8	3	3.2	2	1.0	5	5.2
Gallbladder	9	22.6	10	17.7	7	9.5	8	9.3
Other Biliary	3	4.5	0	0.0	0	0.0	1	0.4
Pancreas	5	9.3	7	10.0	9	11.7	13	12.6
Respiratory	9	15.2	15	21.6	29	35.2	80	78.8*
Lung & Bronchus	9	15.2	14	20.1	29	35.2	75	74.8*
Larynx	0	0.0	0	0.0	0	0.0	4	3.0
Bones & Joints	0	0.0	2	2.6	2	1.0	4	3.4
Soft Tissue	0	0.0	1	2.1	2	2.5	2	2.1
Melanoma of Skin	1	1.2	1	1.5	1	1.2	2	2.1
Breast	26	37.9	42	50.3	49	50.4	92	75.6*
Female Genital	24	29.1	40	47.5	58	51.4	53	36.7
Cervix uteri	12	11.5	27	32.9	42	35.9	28	16.5
Corpus uteri/Uterus, NOS	3	3.4	3	2.7	5	4.1	7	6.8
Ovary	8	11.4	9	9.7	8	9.4	15	11.4
Urinary	11	15.4	12	16.1	12	15.7	26	25.9
Bladder	2	2.0	2	4.2	3	4.5	3	3.2
Kidney & Renal Pelvis	9	13.5	10	11.9	8	10.1	23	22.7
Eye & Orbit	1	0.5	0	0.0	1	0.5	1	0.4
Brain & Nervous System	4	3.0	2	1.0	2	1.7	4	2.0
Brain	3	1.6	2	1.0	2	1.7	4	2.0
Endocrine	10	8.5	8	7.3	8	5.2	11	8.3
Thyroid	10	8.5	8	7.3	8	5.2	11	8.3
Lymphomas	2	5.5	4	4.6	5	5.8	7	5.3
Hodgkin's	0	0.0	0	0.0	2	2.1	2	1.4
Non-Hodgkin's	2	5.5	4	4.6	3	3.7	5	3.9
Multiple Myeloma	2	3.3	0	0.0	1	1.1	2	1.9
Leukemia	3	5.3	3	2.3	3	2.6	5	4.0
Ill Defined & Unspecified	14	31.2	26	44.0	23	32.3	14	16.0

+Rates are age-adjusted to 1970 US standard population.

\*Mantel-Haenszel chi-square for trend,  $p < 0.05$ .



**Table 3. Invasive Cancers in Alaska Natives 1969-1988**  
**Average Annual Age-Specific Incidence Rates per 100,000**  
**Males**

Site	Age							
	0-9	10-19	20-29	30-39	40-49	50-59	60-69	70+
All Sites	15.7	13.8	25.8	57.7	185.8	554.7	1337.3	2314.9
Oral Cavity & Pharynx	-	-	1.7	5.3	34.3	55.0	61.9	37.3
Salivary Gland	-	-	0.8	-	1.8	5.0	4.1	-
Nasopharynx	-	-	-	3.9	21.6	32.5	49.5	18.7
Gum & Other Oral Cavity	-	-	0.8	1.3	3.6	7.5	-	-
Digestive	1.4	4.4	7.5	15.7	70.3	184.9	478.8	927.2
Esophagus	-	-	-	-	3.6	5.0	24.7	93.3
Stomach	-	-	1.7	7.9	14.4	40.0	99.1	149.4
Colon & Rectum	-	0.6	1.7	3.9	25.3	90.0	222.9	516.5
Colon	-	-	1.7	2.6	14.4	62.5	156.8	367.1
Rectosigmoid Junction	-	-	-	1.3	7.2	10.0	12.4	31.1
Rectum	-	0.6	-	-	3.6	17.5	53.7	118.2
Liver	1.4	3.8	4.2	2.6	10.8	20.0	37.2	31.1
Gallbladder	-	-	-	-	-	2.5	8.3	43.6
Other Biliary	-	-	-	-	-	5.0	16.5	24.9
Pancreas	-	-	-	1.3	12.6	20.0	49.5	49.8
Respiratory	-	-	-	5.3	28.9	177.4	421.0	466.7
Lung & Bronchus	-	-	-	2.6	19.8	164.9	412.8	454.3
Larynx	-	-	-	-	5.4	7.5	-	6.2
Bones & Joints	0.7	-	0.8	2.6	1.8	2.5	12.4	6.2
Soft Tissue	-	1.3	-	2.6	-	7.5	-	18.7
Melanoma of Skin	-	-	-	1.3	1.8	-	-	0.0
Breast	-	-	-	-	-	-	4.1	-
Male Genital	2.1	-	10.8	7.9	9.0	30.0	132.1	354.7
Prostate	-	-	-	-	1.8	20.0	128.0	348.5
Testis	0.7	-	10.8	7.9	5.4	5.0	-	-
Urinary	0.7	-	-	2.6	9.0	32.5	99.1	168.0
Bladder	-	-	-	2.6	1.8	7.5	33.0	68.5
Kidney & Renal Pelvis	0.7	-	-	-	7.2	25.0	66.0	93.3
Eye & Orbit	1.4	-	-	-	-	2.5	-	-
Brain & Nervous System	3.4	1.3	1.7	1.3	5.4	-	4.1	6.2
Brain	3.4	1.3	1.7	1.3	5.4	-	4.1	6.2
Endocrine	-	0.6	-	2.6	3.6	12.5	4.1	18.7
Thyroid	-	-	-	2.6	1.8	7.5	-	18.7
Lymphomas	2.1	1.3	1.7	2.6	9.0	7.5	24.8	80.9
Hodgkin's	0.7	-	0.8	1.3	1.8	-	-	-
Non-Hodgkin's	1.4	1.3	0.8	1.3	7.2	7.5	24.8	80.9
Multiple Myeloma	-	-	-	-	1.8	7.5	12.4	37.3
Leukemia	3.4	4.4	1.7	3.9	1.8	2.5	4.1	31.1
Ill Defined and Unspecified	0.7	0.6	-	3.9	7.2	32.5	78.4	149.4

**Table 4. Invasive Cancers in Alaska Natives 1969-1988**  
**Average Annual Age-Specific Incidence Rates per 100,000**  
**Females**

Site	Age							
	0-9	10-19	20-29	30-39	40-49	50-59	60-69	70+
All Sites	10.8	12.5	49.3	115.2	333.8	642.4	1021.4	2024.0
Oral Cavity & Pharynx	-	-	0.8	5.3	26.6	18.5	81.4	49.5
Salivary Gland	-	-	0.8	5.3	5.7	5.3	4.1	6.2
Nasopharynx	-	-	-	-	13.3	7.9	48.8	-
Gum & Other Oral Cavity	-	-	-	-	1.9	-	8.1	24.8
Digestive	-	2.6	5.0	11.9	45.5	163.9	419.1	940.8
Esophagus	-	-	-	-	-	10.6	12.2	31.0
Stomach	-	0.7	2.5	4.0	9.5	7.9	16.3	68.1
Colon & Rectum	-	-	1.7	4.0	28.5	97.8	260.4	600.4
Colon	-	-	1.7	2.7	22.8	74.0	215.7	415.8
Rectosigmoid Junction	-	-	-	-	3.8	2.6	16.3	55.7
Rectum	-	-	-	1.3	1.9	21.2	28.5	92.8
Liver	-	2.0	0.8	-	-	2.6	8.1	24.8
Gallbladder	-	-	-	-	-	10.6	40.7	123.8
Other Biliary	-	-	-	1.3	1.9	2.6	4.1	-
Pancreas	-	-	-	1.3	3.8	23.8	61.0	43.3
Respiratory	-	-	0.8	6.6	37.9	76.7	166.8	229.0
Lung & Bronchus	-	-	0.8	5.3	36.0	74.0	154.6	229.0
Larynx	-	-	-	1.3	1.9	2.6	4.1	-
Bones & Joints	0.7	1.3	0.8	-	-	5.3	-	12.4
Soft Tissue	-	-	-	-	1.9	2.6	-	18.6
Melanoma of Skin	-	-	-	-	1.9	2.6	8.1	6.2
Breast	-	-	5.0	30.5	113.8	163.9	118.0	179.5
Female Genital	0.7	2.6	21.7	43.7	58.8	108.4	89.5	105.2
Cervix uteri	-	1.3	13.4	37.1	43.6	50.2	52.9	49.5
Corpus uteri/Uterus, NOS	-	-	1.7	2.7	5.7	18.5	12.2	6.2
Ovary	0.7	1.3	5.0	1.3	9.5	37.0	24.4	40.0
Urinary	1.4	0.7	0.8	4.0	17.1	34.4	40.7	136.2
Bladder	-	-	-	1.3	3.8	-	4.1	37.1
Kidney & Renal Pelvis	1.4	0.7	0.8	2.7	13.3	31.7	36.6	99.0
Eye & Orbit	2.2	-	-	-	-	-	-	-
Brain & Nervous System	3.6	2.0	-	1.3	-	5.3	4.1	-
Brain	3.6	2.0	-	1.3	-	2.6	4.1	-
Endocrine	-	0.7	9.2	9.3	22.8	5.3	8.1	12.4
Thyroid	-	0.7	9.2	9.3	22.8	5.3	8.1	12.4
Lymphomas	-	0.7	2.5	-	7.6	5.3	12.2	31.0
Hodgkin's	-	-	1.7	-	-	-	4.1	6.2
Non-Hodgkin's	-	0.7	0.8	-	7.6	5.3	8.1	24.8
Multiple Myeloma	-	-	-	-	-	7.9	8.1	-
Leukemia	1.4	1.3	1.7	1.3	-	5.3	8.1	18.6
Ill Defined & Unspecified	0.7	0.7	0.8	1.3	-	37.0	57.0	278.5



**Table 5. Invasive Cancers in Alaska Natives 1969-1988**  
**Average Annual Incidence Rates per 100,000**  
**by Ethnicity, Males**

Site	Numbers and Age-Adjusted* Rates					
	Aleut		Indian		Eskimo	
	No.	Rate	No.	Rate	No.	Rate
All Sites	158	330.3	395	326.6	585	302.4
Oral Cavity & Pharynx	14	24.6	18	14.0	36	16.8
Salivary Gland	1	2.2	1	0.8	3	1.2
Nasopharynx	10	17.8	6	4.7	27	13.1
Gum & Other Oral Cavity	0	0.0	4	2.8	3	1.1
Digestive	51	111.4	110	93.2	247	130.2
Esophagus	4	8.8	9	7.9	12	7.0
Stomach	11	19.0	11	9.7	58	30.6
Colon & Rectum	28	63.7	67	57.6	98	54.1
Colon	20	43.7	44	38.1	70	39.3
Rectosigmoid Junction	2	3.2	8	6.3	7	3.6
Rectum	6	16.8	15	13.1	21	11.3
Liver	1	2.2	6	4.0	36	15.1
Gallbladder	0	0.0	3	2.7	7	4.1
Other Biliary	1	1.7	3	2.7	6	3.3
Pancreas	6	16.1	9	7.0	21	11.0
Respiratory	37	75.2	81	71.9	150	80.9
Lung & Bronchus	32	67.1	76	68.1	144	78.2
Larynx	1	1.7	3	2.3	3	1.4
Bones & Joints	2	2.8	3	2.4	5	1.9
Soft Tissue	1	1.0	5	4.3	4	1.3
Melanoma of Skin	0	0.0	1	0.6	1	0.3
Breast	0	0.0	1	0.9	0	0.0
Male Genital	17	36.9	75	59.3	36	19.1
Prostate	13	31.2	57	51.5	26	15.5
Testis	3	5.3	6	6.6	16	1.8
Urinary	9	21.9	31	27.0	32	17.1
Bladder	3	8.7	11	9.7	11	5.8
Kidney & Renal Pelvis	5	10.0	20	17.3	21	11.3
Eye & Orbit	0	0.0	1	0.4	2	0.7
Brain & Nervous System	2	2.1	6	3.3	7	2.2
Brain	2	2.1	6	3.3	7	2.2
Endocrine	1	1.7	9	6.3	4	1.9
Thyroid	0	0.0	7	5.2	2	0.9
Lymphomas	8	18.6	11	8.2	17	7.8
Hodgkin's	1	1.5	0	0.0	3	0.8
Non-Hodgkin's	7	17.1	11	8.2	14	7.0
Multiple Myeloma	1	3.2	9	7.5	3	1.7
Leukemia	4	6.4	11	7.5	10	2.4
Ill Defined & Unspecified	11	24.5	23	18.7	31	17.1

\* Rates are age-adjusted to 1970 U.S. standard population.

**Table 6. Invasive Cancers in Alaska Natives 1969-1988**  
**Average Annual Incidence Rates per 100,000**  
**by Ethnicity, Females**

Site	Numbers and Age-Adjusted* Rates					
	Aleut		Indian		Eskimo	
	No.	Rate	No.	Rate	No.	Rate
All Sites	187	454.3	433	365.1	556	344.1
Oral Cavity & Pharynx	7	17.3	18	14.7	29	16.8
Salivary Gland	0	0.0	5	3.2	7	3.3
Nasopharynx	3	6.5	4	3.4	15	8.4
Gum & Other Oral Cavity	2	6.5	2	1.8	3	2.5
Digestive	63	174.3	98	94.8	199	143.3
Esophagus	1	2.0	1	0.8	10	7.7
Stomach	3	7.5	12	9.7	15	9.5
Colon & Rectum	42	124.3	58	57.9	118	86.2
Colon	31	90.3	41	41.2	98	71.0
Rectosigmoid Junction	4	11.3	4	3.8	8	6.7
Rectum	7	22.8	13	12.9	12	8.5
Liver	1	2.4	4	4.5	6	2.4
Gallbladder	1	4.1	9	9.7	24	18.9
Other Biliary	0	0.0	1	0.8	3	1.4
Pancreas	12	26.9	7	6.4	15	10.4
Respiratory	29	71.3	46	42.0	58	38.7
Lung & Bronchus	26	65.8	45	41.4	56	37.5
Larynx	2	3.1	1	0.6	1	0.6
Bones & Joints	1	2.0	3	2.7	4	1.2
Soft Tissue	0	0.0	1	1.2	4	2.9
Melanoma of Skin	1	1.7	0	0.0	4	2.8
Breast	26	54.9	105	82.1	78	41.8
Female Genital	27	55.2	72	48.5	76	34.8
Cervix uteri	18	33.7	39	24.1	52	23.4
Corpus uteri/Uterus, NOS	1	2.0	11	7.6	6	3.3
Ovary	7	15.3	18	13.3	15	7.3
Urinary	10	27.7	24	21.6	27	17.3
Bladder	5	16.4	3	2.7	2	1.4
Kidney & Renal Pelvis	5	11.3	20	18.1	25	15.9
Eye & Orbit	1	1.1	2	0.8	0	0.0
Brain & Nervous System	2	3.5	2	1.2	8	2.2
Brain	2	3.5	1	0.4	8	2.2
Endocrine	6	10.4	17	9.9	14	5.2
Thyroid	6	10.4	17	9.9	14	5.2
Lymphomas	4	9.2	7	6.6	7	3.3
Hodgkin's	1	4.1	1	1.0	2	0.5
Non-Hodgkin's	3	5.1	6	5.6	5	2.8
Multiple Myeloma	0	0.0	4	3.5	1	0.5
Leukemia	2	4.4	5	4.5	7	2.4
Ill Defined & Unspecified	8	21.4	29	31.2	40	30.2

\* Rates are age-adjusted to 1970 U.S. standard population.



**Table 7. Invasive Cancers in Alaska Natives 1984-1988**  
**Ratio of Alaska Native to SEER Average Annual Age-Adjusted\* Incidence Rates per 100,000**

Site	Males			Females		
	ALASKA No.	Rate	AK/SEER Rate Ratio	ALASKA No.	Rate	AK/SEER Rate Ratio
All Sites	372	332.7	0.8*	453	414.5	1.2*
Oral Cavity & Pharynx	23	19.1	1.2	14	13.4	2.1
Salivary Gland	1	1.0	0.8	3	2.1	2.6
Nasopharynx	11	8.9	14.8*	6	5.6	18.7*
Gum & Other Oral Cavity	3	2.1	0.8	1	1.2	0.8
Digestive	132	118.9	1.2*	136	137.6	2.2*
Esophagus	7	7.0	1.3	5	5.3	3.3
Stomach	26	22.2	2.1*	14	12.0	2.7*
Colon & Rectum	66	61.9	1.0	86	89.3	2.1*
Liver	11	7.7	2.7*	5	5.2	4.3
Gallbladder	1	1.0	1.3	8	9.3	6.6*
Other Biliary	7	6.9	4.9*	1	0.4	0.4
Pancreas	12	10.3	1.0	13	12.6	1.6
Respiratory	102	95.5	1.0	80	78.8	2.0*
Lung & Bronchus	97	91.4	1.1	75	74.8	2.0*
Larynx	1	0.8	0.1*	4	3.0	1.9
Bones & Joints	5	3.1	3.1	4	3.4	4.9
Soft Tissue	2	1.7	0.7	2	2.1	1.2
Melanoma of Skin	0	0.0	-	2	2.1	0.2*
Breast	0	0.0	-	92	75.6	0.7*
Female Genital	0	0.0	-	53	36.7	0.8*
Cervix uteri	0	0.0	-	28	16.5	2.1*
Corpus uteri/Uterus, NOS	0	0.0	-	7	6.8	0.3*
Ovary	0	0.0	-	15	11.4	0.8
Male Genital	41	36.3	0.4*	0	0.0	-
Prostate	31	31.1	0.3*	0	0.0	-
Testis	8	3.7	0.8	0	0.0	-
Urinary	27	25.4	0.6*	26	25.9	1.9*
Bladder	7	6.2	0.2*	3	3.2	0.4*
Kidney & Renal Pelvis	20	19.1	1.6	23	22.7	4.1*
Eye & Orbit	0	0.0	-	1	0.4	0.7
Brain & Nervous System	4	2.2	0.3*	4	2.0	0.4*
Brain	4	2.2	0.3*	4	2.0	0.4*
Endocrine	4	2.8	0.9	11	8.3	1.3
Thyroid	2	1.5	0.6	11	8.3	1.4
Lymphomas	9	7.5	0.4*	7	5.3	0.4*
Hodgkin's	1	0.4	0.1*	2	1.4	0.5
Non-Hodgkin's	8	7.1	0.4*	5	3.9	0.3*
Multiple Myeloma	2	2.0	0.4	2	1.9	0.6
Leukemia	8	4.7	0.4*	5	4.0	0.5*
Ill Defined & Unspecified	13	10.8	0.9	14	16.0	1.8

+Alaska Native and SEER rates are age-adjusted to 1970 US standard population.

\* The difference between Alaska Native and Seer age-adjusted incidence rates are statistically significant at the 95% confidence level.

**Table 8. Invasive Cancers in Alaska Natives 1984-1988**  
**Ratio of Alaska Native to SEER Average Annual Age-Adjusted Incidence Rates<sup>+</sup> per 100,000**

Males									
Site	Aleut			Indian			Eskimo		
	No.	Rate	Rate Ratio	No.	Rate	Rate Ratio	No.	Rate	Rate Ratio
Lung/Bronchus	13	87.5	1.1	28	77.9	0.9	56	101.7	1.2
Colon/Rectum	14	114.2	1.9	17	44.0	0.7	35	63.1	1.0
Prostate	6	43.2	0.5*	17	48.6	0.5*	8	15.5	0.2*
Females									
Site	Aleut			Indian			Eskimo		
	No.	Rate	Rate Ratio	No.	Rate	Rate Ratio	No.	Rate	Rate Ratio
Lung/Bronchus	15	121.3	3.2	29	76.7	2.0	31	62.4	1.7*
Colon/Rectum	16	135.9	3.2	21	60.6	1.4	49	98.9	2.3*
Cervix	1	7.7	1.0	11	15.3	2.0	16	19.5	2.5*
Breast	9	66.9	0.6	54	116.7	1.2	29	47.1	0.4*

\* Rates are age-adjusted to 1970 US standard population.

\* The difference between Alaska Native and Seer age-adjusted incidence rates are statistically significant at the 95% confidence level.

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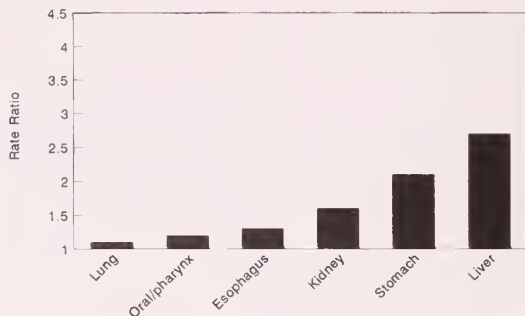
# COMPARISON OF CANCER PATTERNS (1984-1988) ALASKA NATIVES vs U.S.

This section includes four graphs. Alaska Native age-adjusted average annual incidence rates are compared to published SEER rates for the U.S. white population. Cancer sites noted on the top graphs are those for which the Alaska Native people experience more cancers of that type than U.S. Whites. Those on the bottom graphs are cancers which occur relatively less frequently among Alaska Natives than U.S. Whites at this time. The height of the bar indicates the magnitude of the difference between cancer rates in Alaska Natives and U.S. Whites. For example Alaska Native women are two times as likely to develop lung cancer as U.S. White women. On the other hand U.S. White women experience more than three times the rate of cancer of the uterus.

Only cancer sites for which there were enough cancers to meaningfully compare rates are noted.

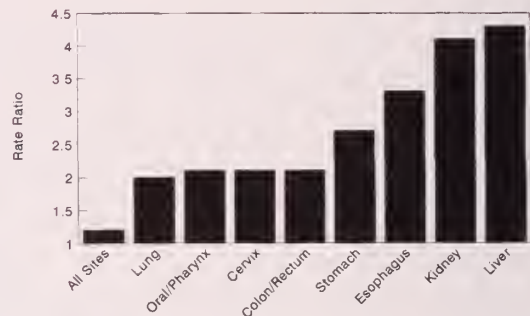
Cancer sites for which the bars are approximately equal to "1" indicate similar rates are occurring in the two populations being compared. Note that for all invasive cancers ("All Sites"), rates are similar in men, but Alaska Native female rates exceed those of U.S. White women. The sites listed in the top graph for females are responsible for this excess. It is important to remember that although the magnitude of the difference is greatest in women for liver and kidney cancer for example, the more frequently diagnosed cancers that occur in excess (lung, colon/rectum, etc.) are most responsible for the excess in all sites of cancer.

Average Annual Age-Adjusted Cancer Incidence Rates 1984-1988  
Alaska Native Rates Greater than U.S. Rates\*  
**Select Cancer Sites: Male**



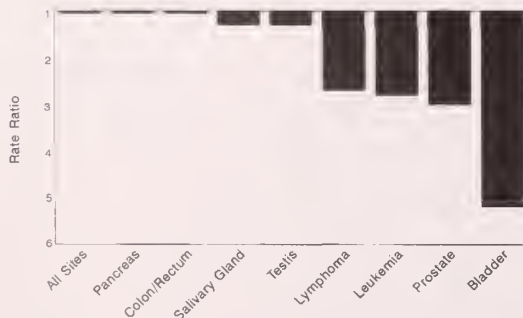
\*Ratio of Alaska Native Rates to US (SEER)

Average Annual Age-Adjusted Cancer Incidence Rates 1984-1988  
Alaska Native Rates Greater than U.S. Rates\*  
**Select Cancer Sites: Female**



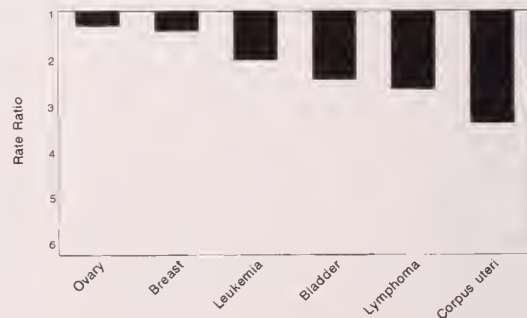
\*Ratio of Alaska Native Rates to US (SEER)

Alaska Native Rates Less than or Equal to U.S. Rates\*  
**Select Cancer Sites: Male**



\*Ratio of US Rates to Alaska Native Rates

Alaska Native Rates Less than U.S. Rates\*  
**Select Cancer Sites: Female**



\*Ratio of US Rates to Alaska Native Rates

## **CANCER INCIDENCE BY CANCER SITE: AGE-SPECIFIC RATES, TRENDS AND REGIONAL DISTRIBUTION (1969 - 1988)**

In this section, data are presented by cancer site (body organ), or site group. Cancers are categorized and enumerated by the part of the body in which the cancer first developed. A unique coding system (International Classification of Disease for Oncology, ICD-O) has been developed and accepted for use worldwide by tumor registries in order to facilitate comparing cancer data from different registries and for different populations. Some cancers occur infrequently, therefore data are often shown by site group. The ICD-O-2 codes, cancer sites, and site groupings we have used are included in the Appendix.

The data are compiled in this section in alphabetic order by cancer site. The term most commonly used by cancer registries is given. Information is presented for: trends in rates over time, age-specific rates, and regional distribution. To show trends over the twenty years 1969-88 rates were calculated for each of four different five-year time periods. Rates are expressed as number of cases per 100,000 population. "Average annual" means that all cases occurring in the five-year time period were added together and divided by five to give the average number per year. "Adjusted" means that the Alaska Native rates have been modified to take into consideration the overall young age of the population. Comparison of rates with those for other populations can be meaningful only if the rates have been adjusted. Rates shown for U.S. Blacks and Whites are from the National Cancer Institute's SEER (Statistics, Epidemiology, and End Results) cancer database (reference #7 - Project

Summary). Published data are available from SEER only since 1974, and for some sites only for the years, 1984-88.

Age-specific rates are shown for ten year age groups. Again rates are shown per 100,000 population; number of cases of cancer that would have occurred in 100,000 people of that age. Rates indicate the risk or chances of getting a particular cancer in a given decade of life. As can be seen, for nearly all cancers the risk goes up dramatically with age especially after age forty.

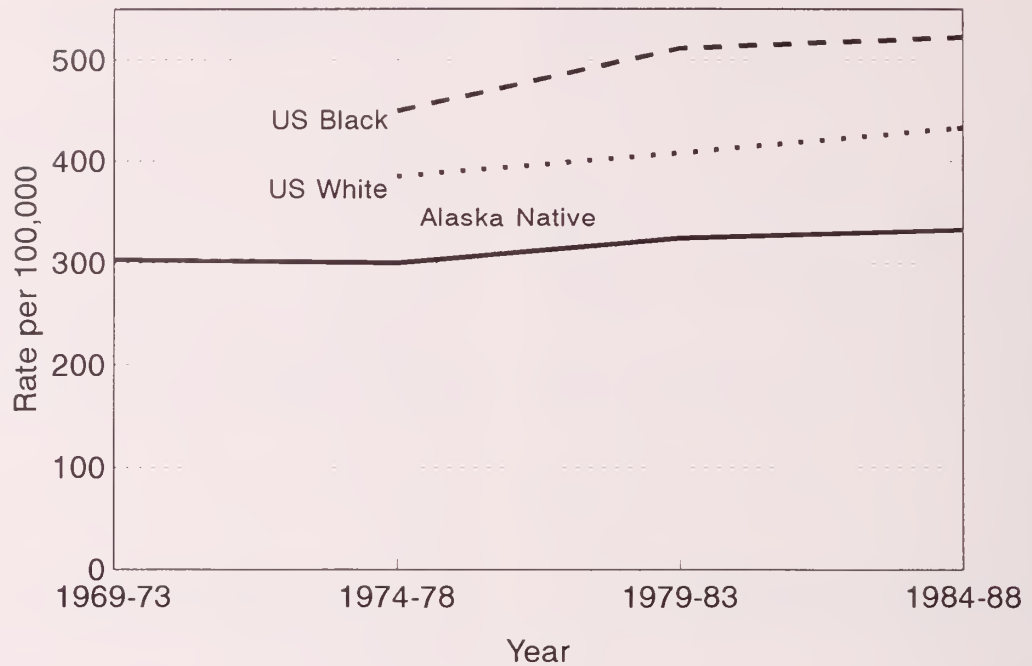
The third set of data includes a map of rates by region based on patients' residence at diagnosis. Rates were calculated when there were five or more cases. Rates calculated on less than five cases may be misleading. When the data are examined by cancer site and separately for males and females, the number of cases in some IHS Service Units can be small to nonexistent. Therefore, we have chosen to combine Service Units and present rates for five regions only. With the exception of Anchorage, these rates largely reflect rates of one ethnic/linguistic group. Again rates are per 100,000, and are displayed for each region and for each sex separately. Rates have been adjusted for age to allow comparison with other U.S. data. For those cancer sites for which there were not enough cases to calculate a meaningful rate for the region, an asterisk (\*) is shown, or the map was omitted entirely.

Number of cases for all cancer sites for each sex and for each of the five service unit groups are listed in the Appendix.

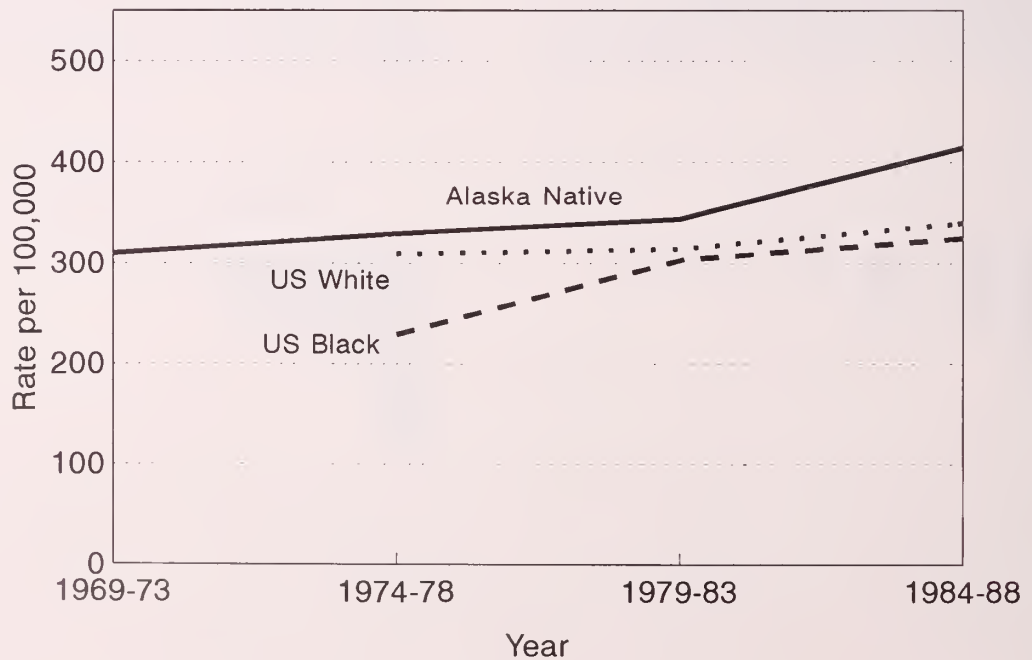


Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US 1974-1988

**All Cancer Sites Combined: Male**



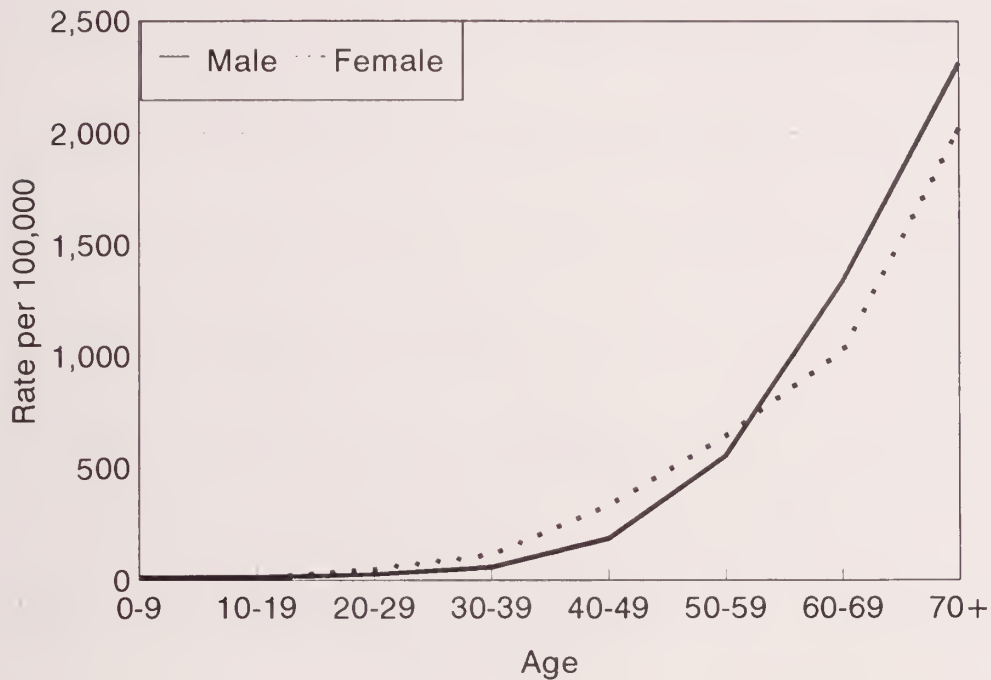
**All Cancer Sites Combined: Female**



<sup>1</sup>US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.

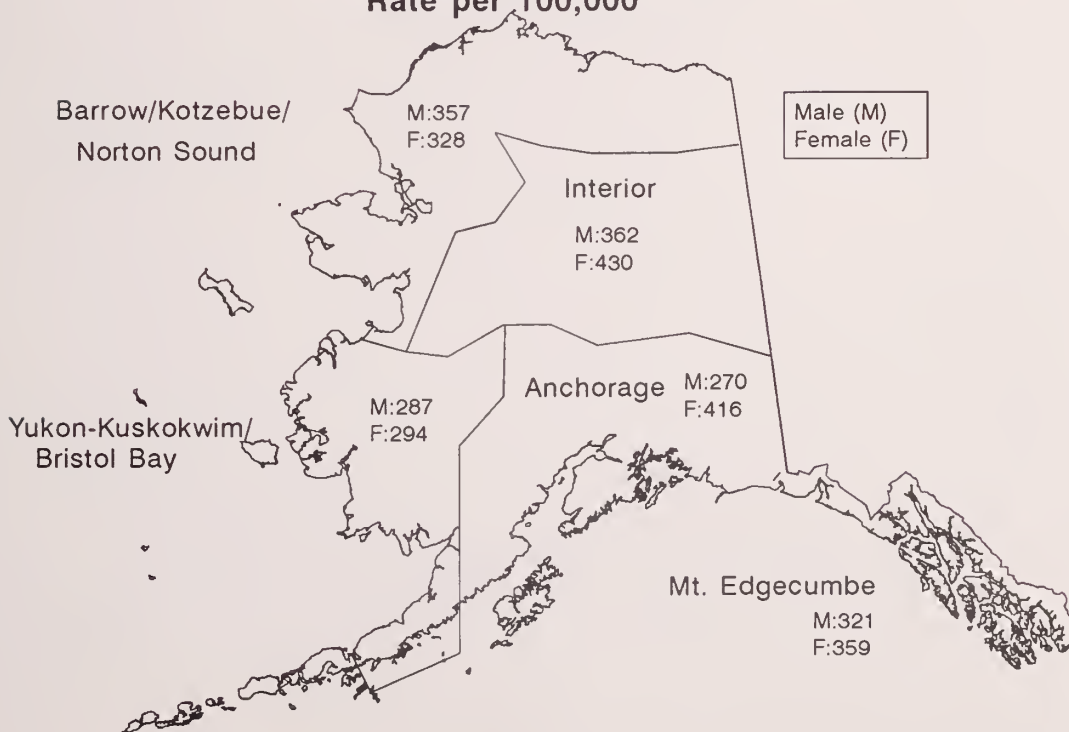
Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

**All Cancer Sites Combined**



Average Annual Age-Adjusted Cancer Incidence 1969-1988

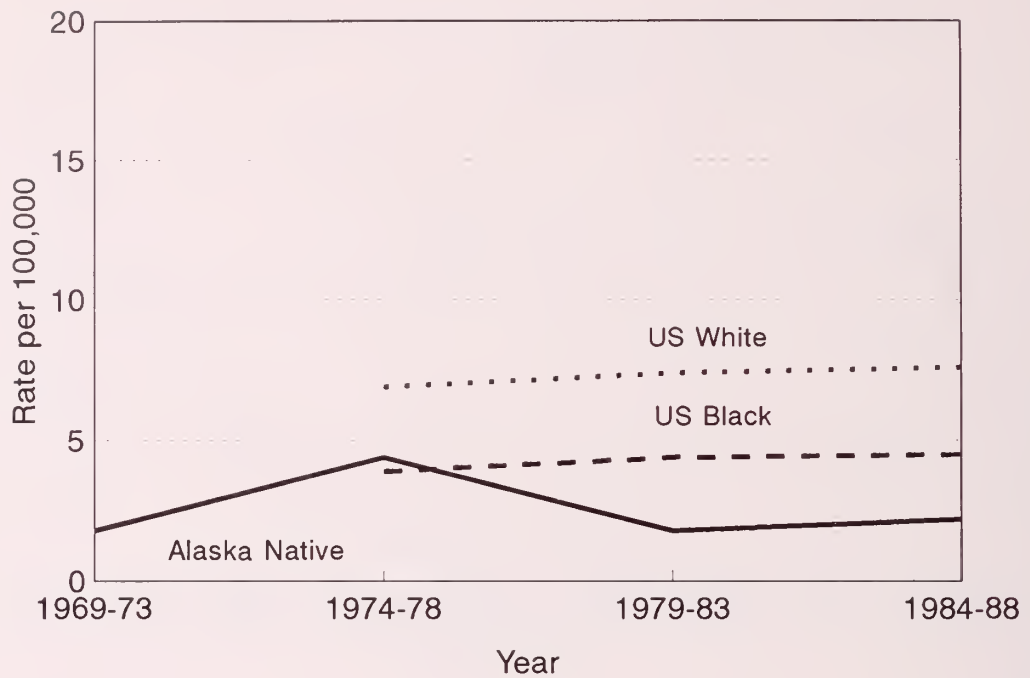
**All Cancer Sites Combined**  
**Rate per 100,000**



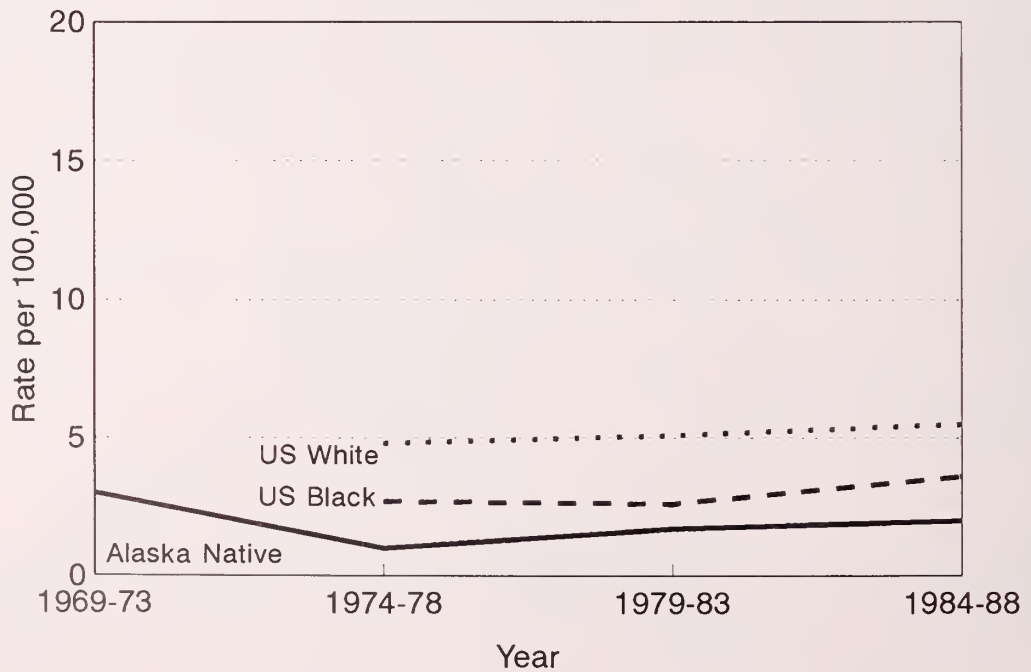


Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US 1974-1988

***Brain and Nervous System: Male***



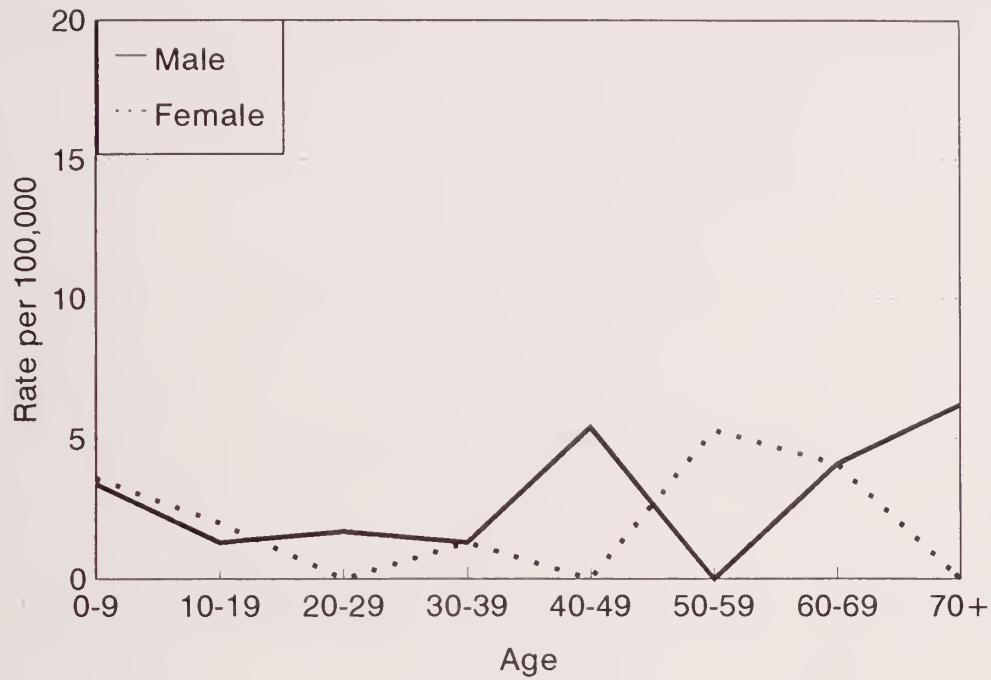
***Brain and Nervous System: Female***



<sup>1</sup>US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.

Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

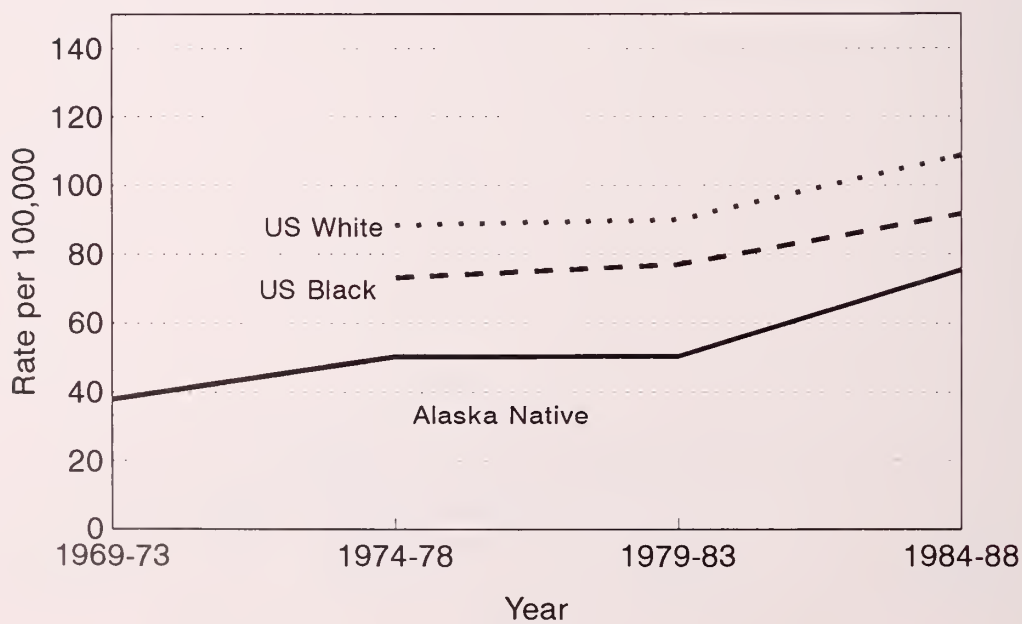
***Brain and Nervous System***



Too few cases to show rates by Service Unit.



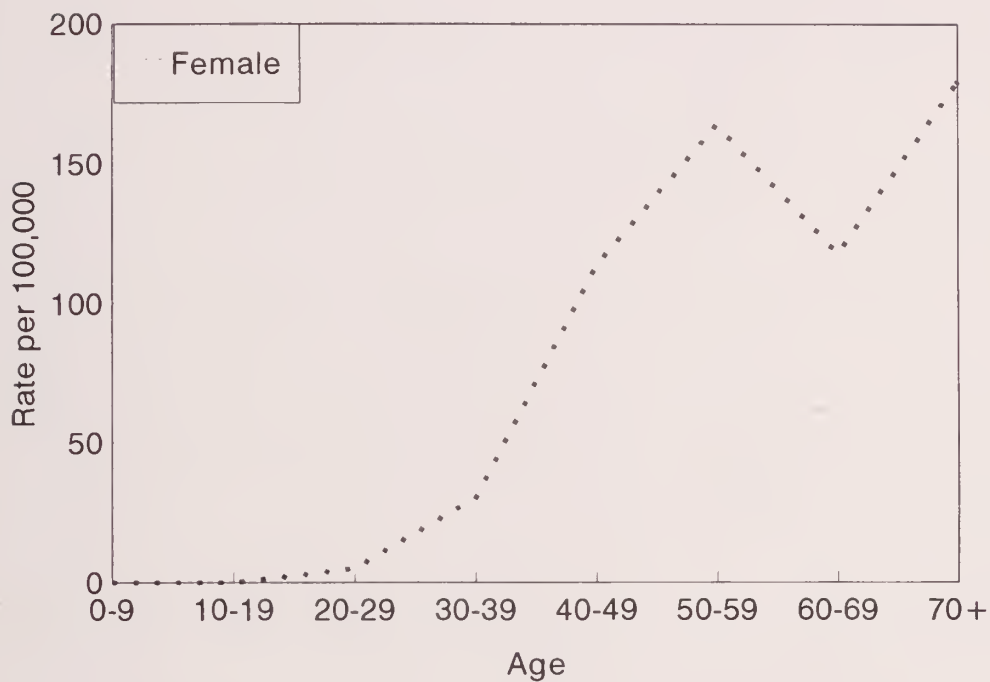
Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US 1974-1988  
**Breast: Female**



<sup>1</sup>US rates from SEER, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.

Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

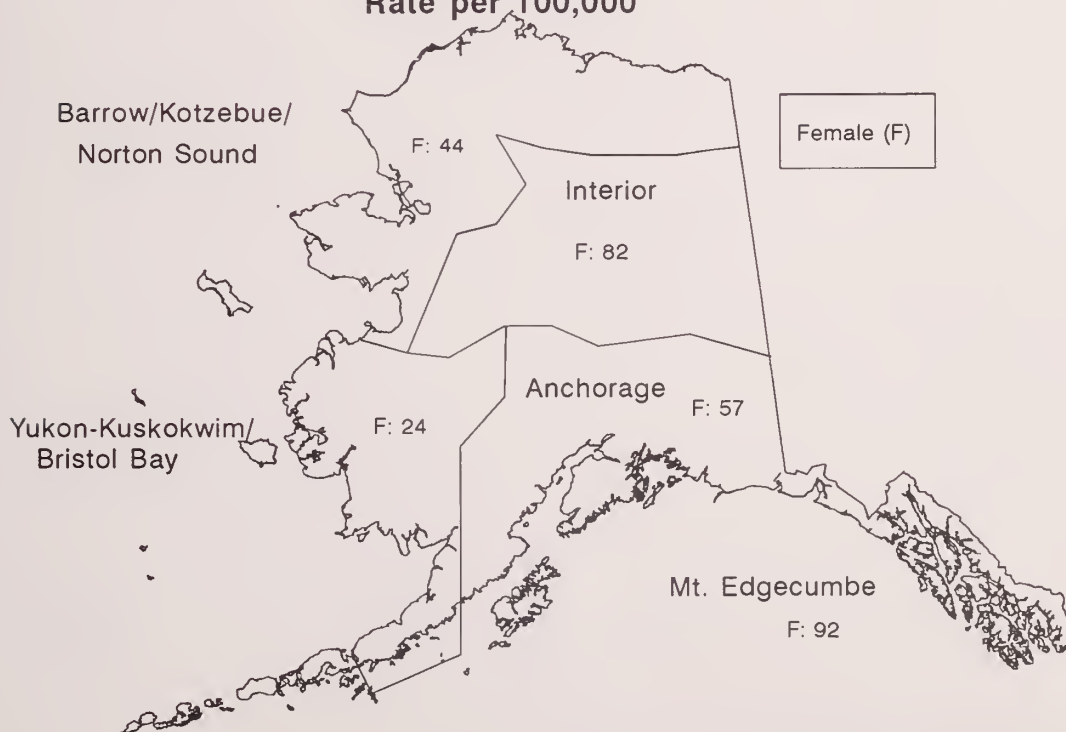
**Breast**



Average Annual Age-Adjusted Cancer Incidence 1969-1988

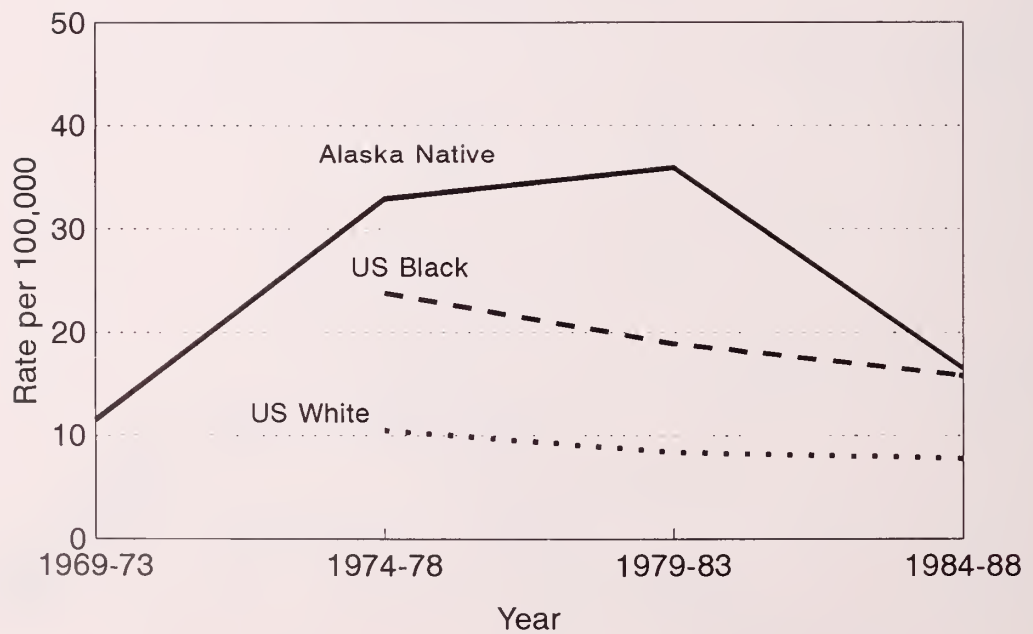
**Breast**

Rate per 100,000





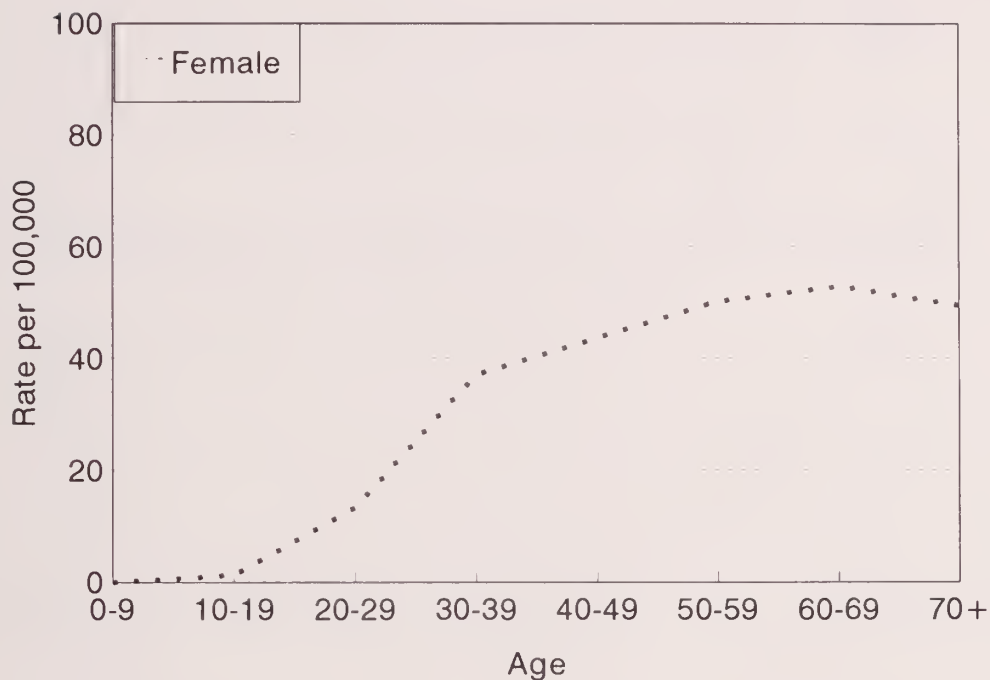
Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US<sup>1</sup> 1974-1988  
**Cervix**



<sup>1</sup>US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.

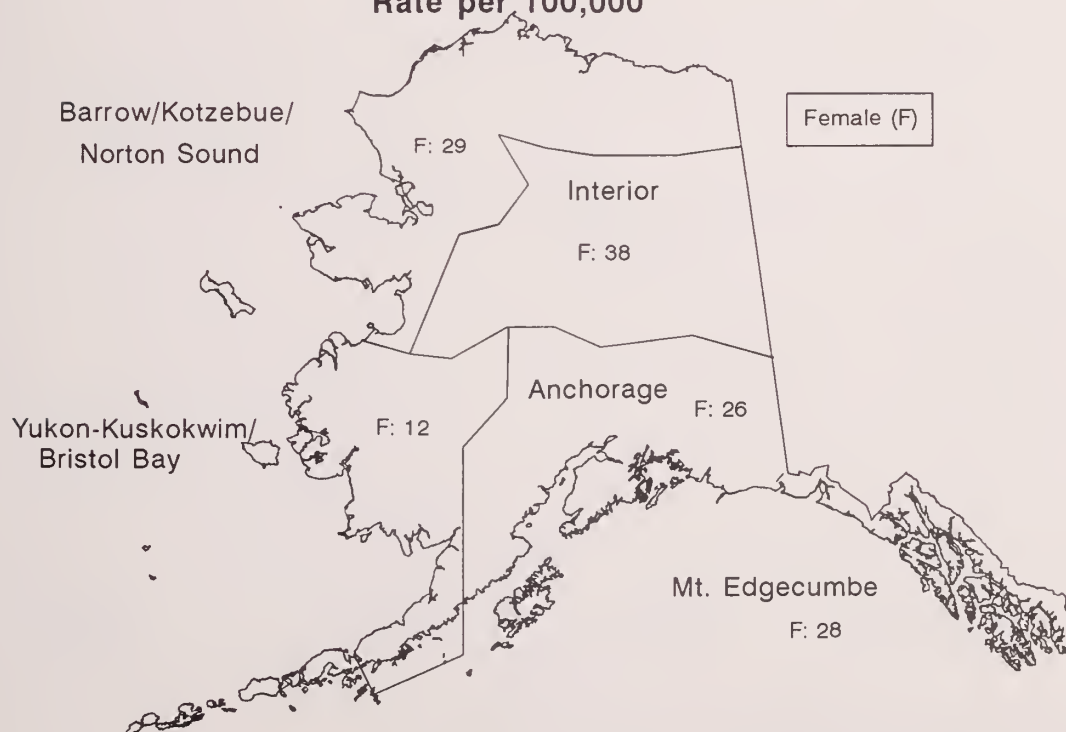
Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

**Cervix**



Average Annual Age-Adjusted Cancer Incidence 1969-1988

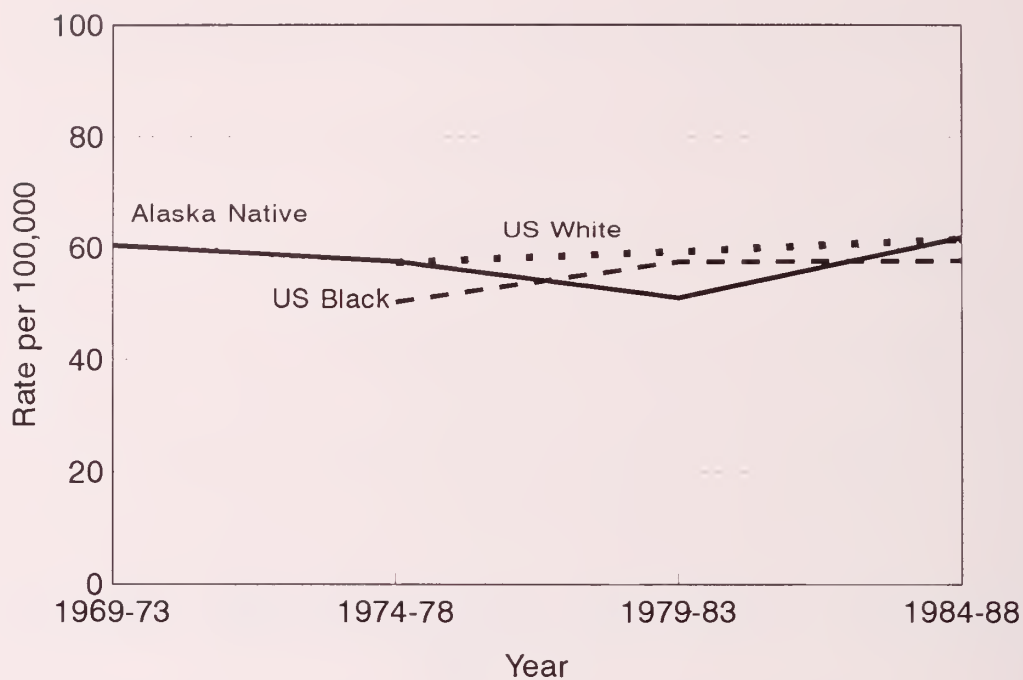
**Cervix**  
**Rate per 100,000**



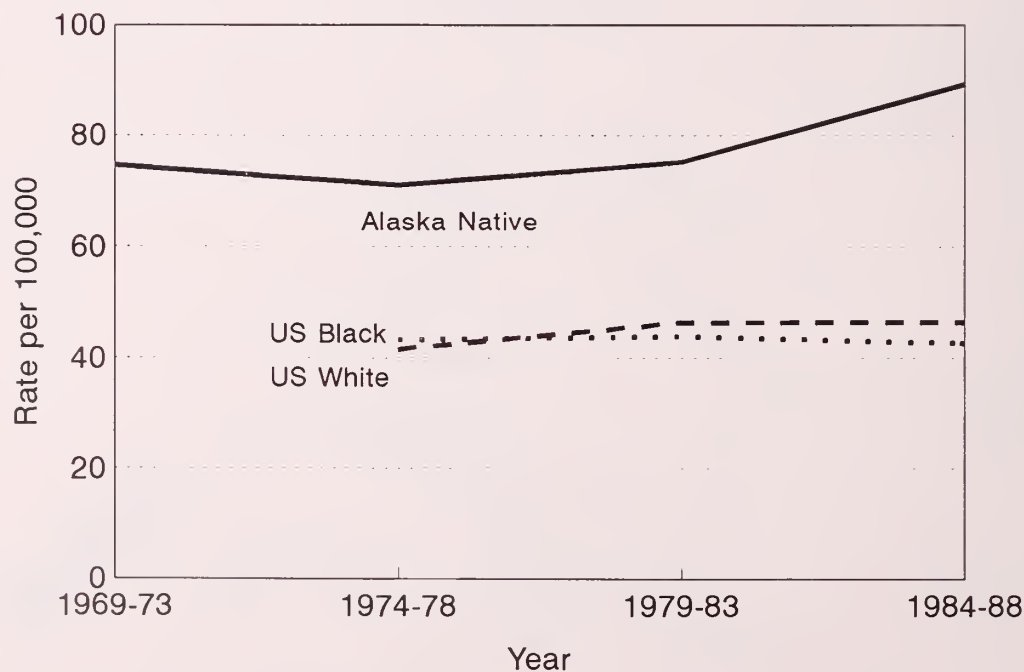


Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US<sup>1</sup> 1974-1988

**Colon/Rectum: Male**



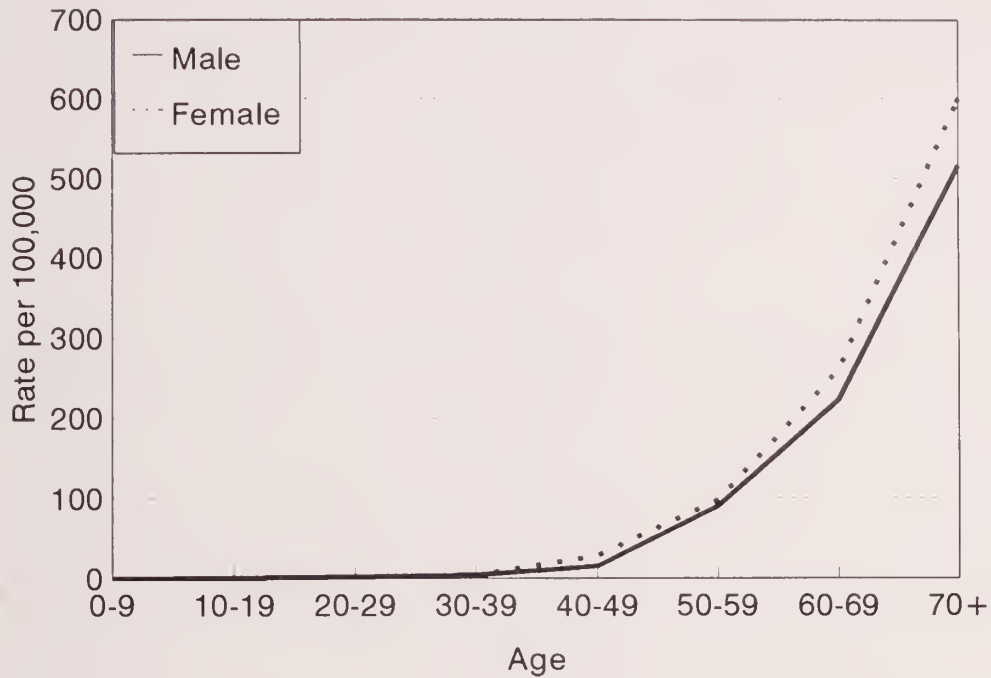
**Colon/Rectum: Female**



<sup>1</sup>US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.

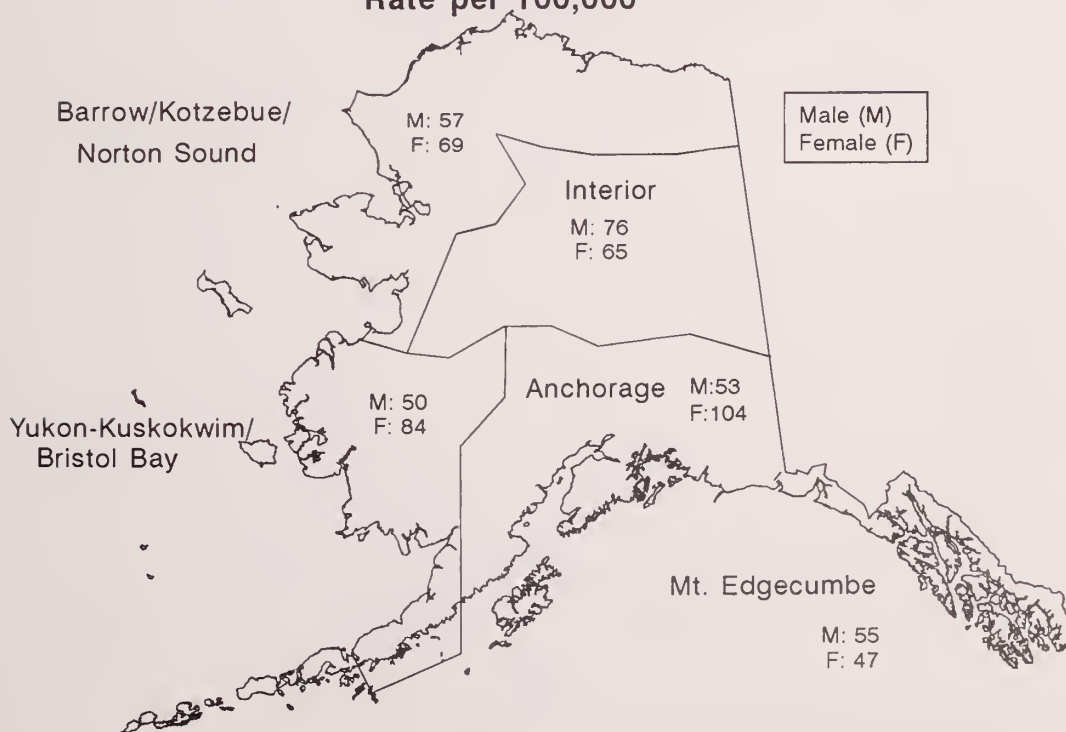
Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

**Colon/Rectum**



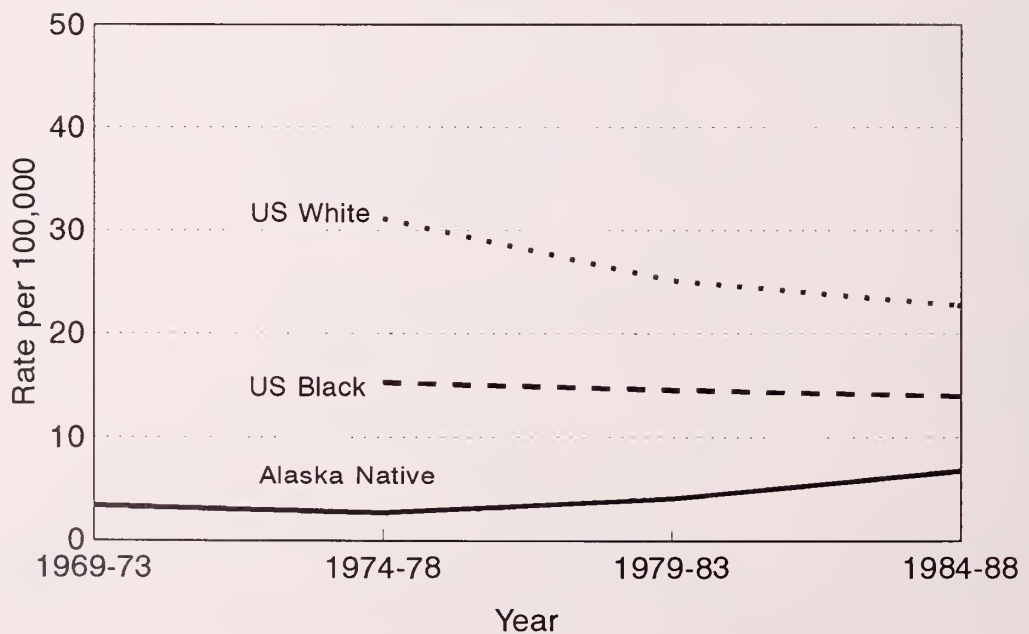
Average Annual Age-Adjusted Cancer Incidence 1969-1988

**Colon/Rectum**  
**Rate per 100,000**





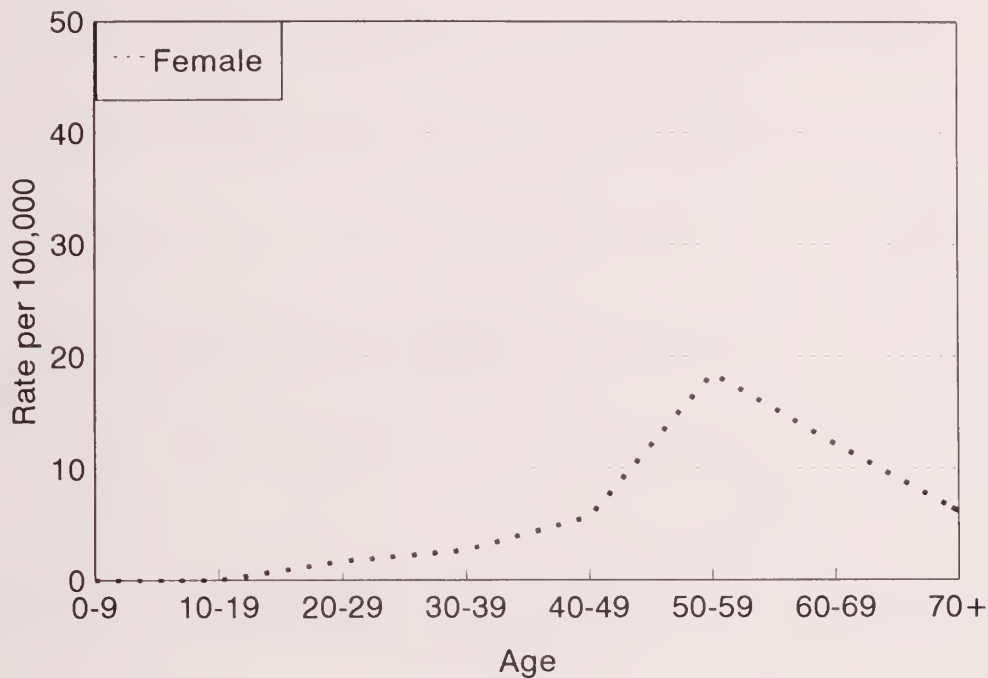
Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US<sup>1</sup> 1974-1988  
***Corpus uteri/Uterus, NOS***



<sup>1</sup>US Rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.

Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

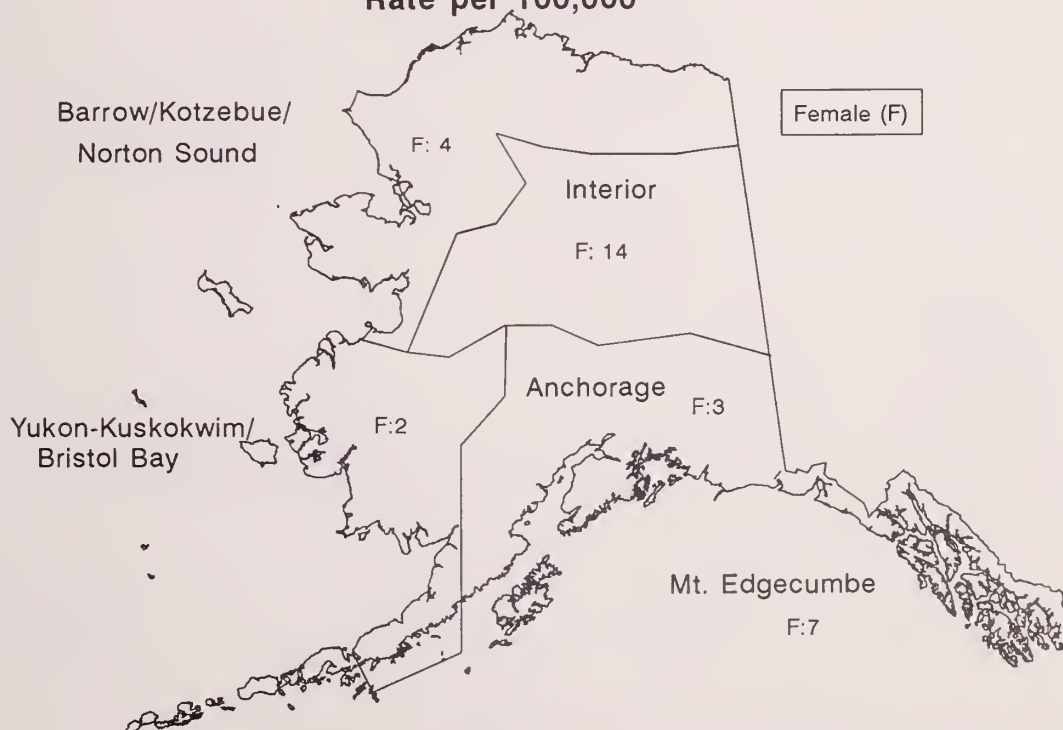
**Corpus uteri/Uterus, NOS**



Average Annual Age-Adjusted Cancer Incidence 1969-1988

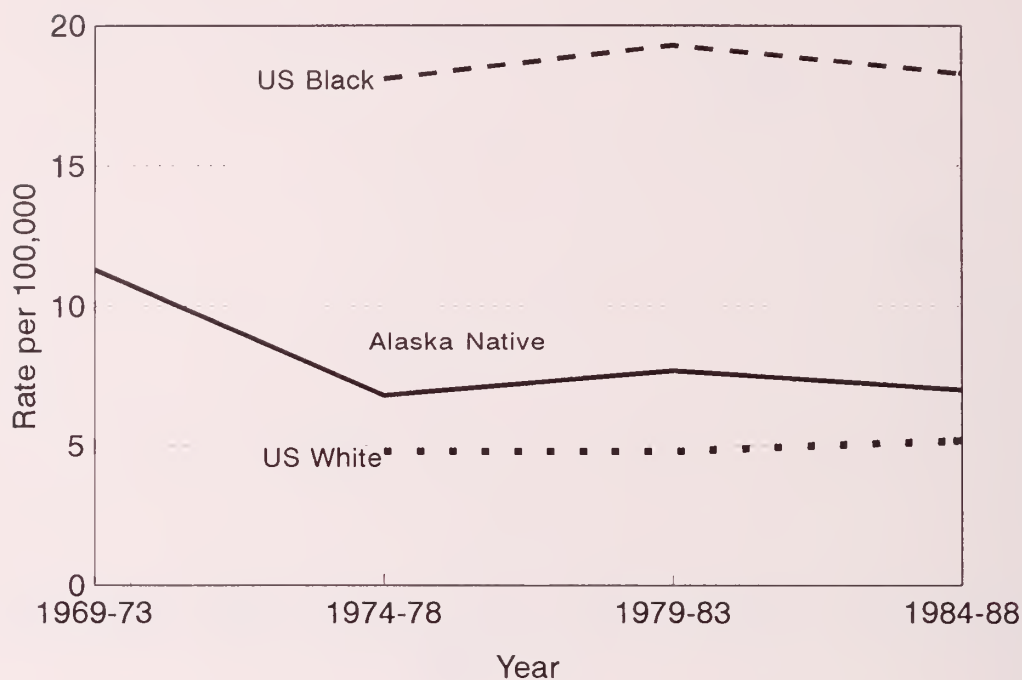
**Corpus uteri/Uterus, NOS**

Rate per 100,000

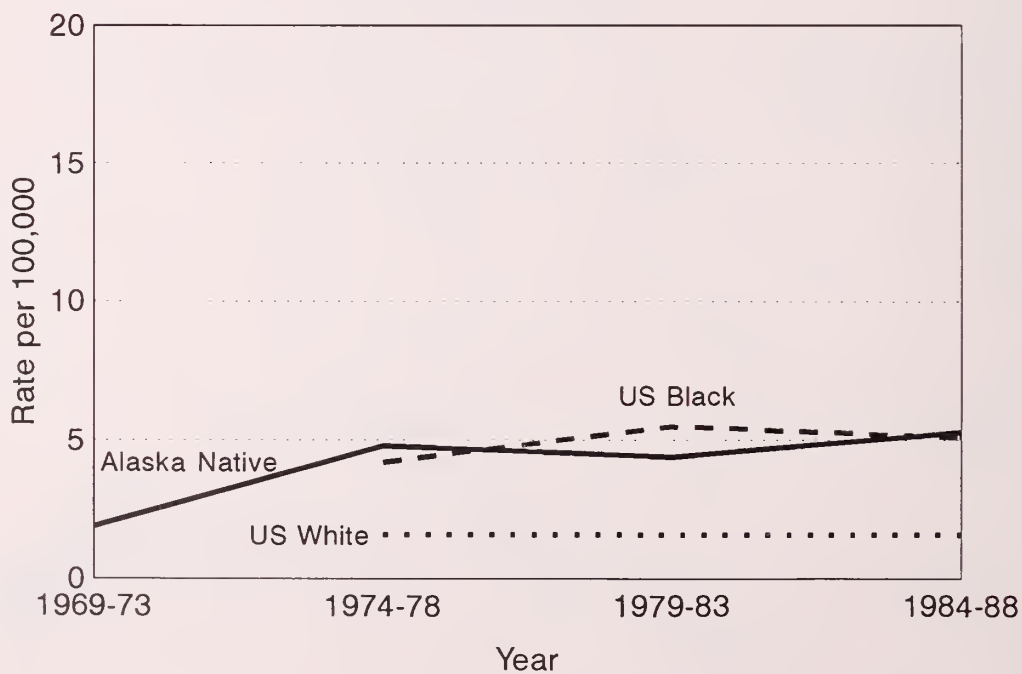


Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US 1974-1988

***Esophagus: Male***



***Esophagus: Female***

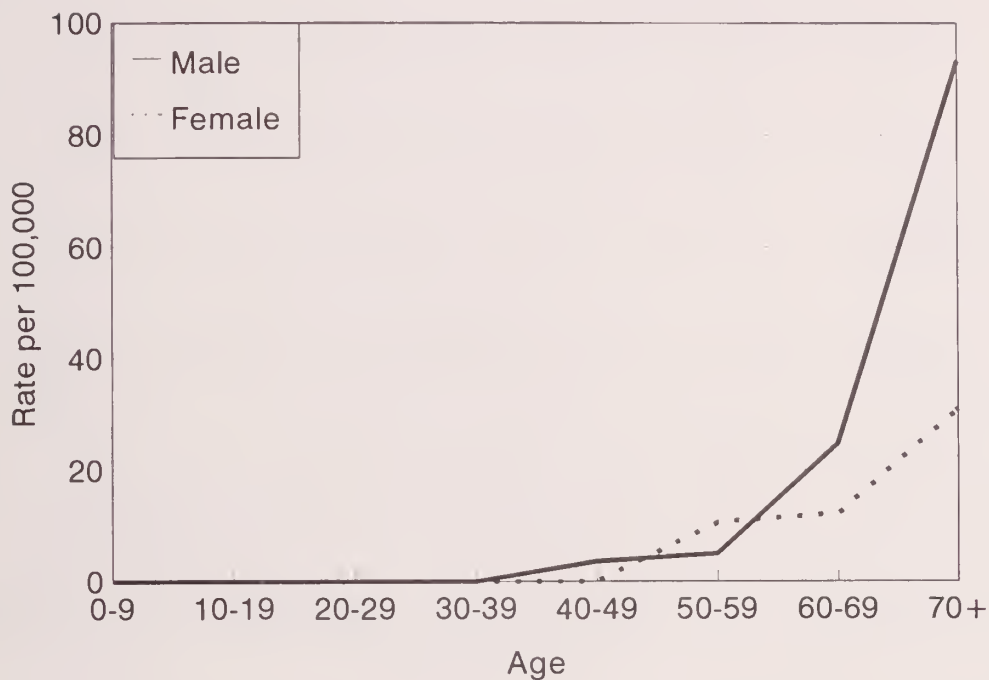


<sup>1</sup>US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.



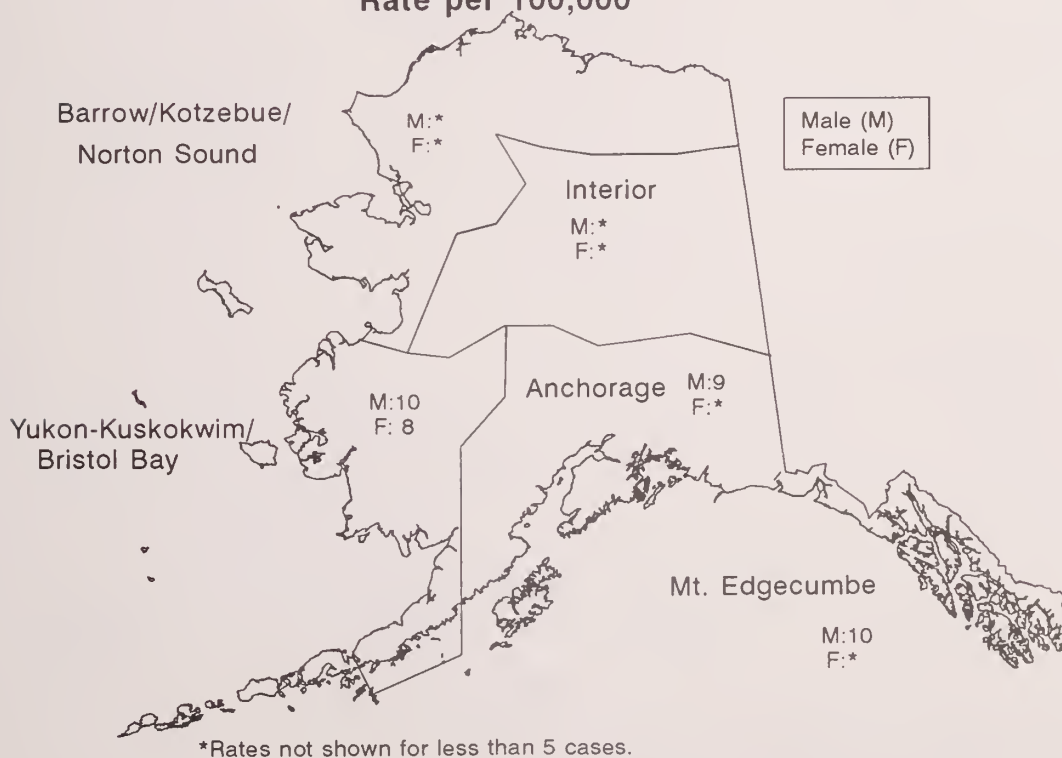
Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

**Esophagus**



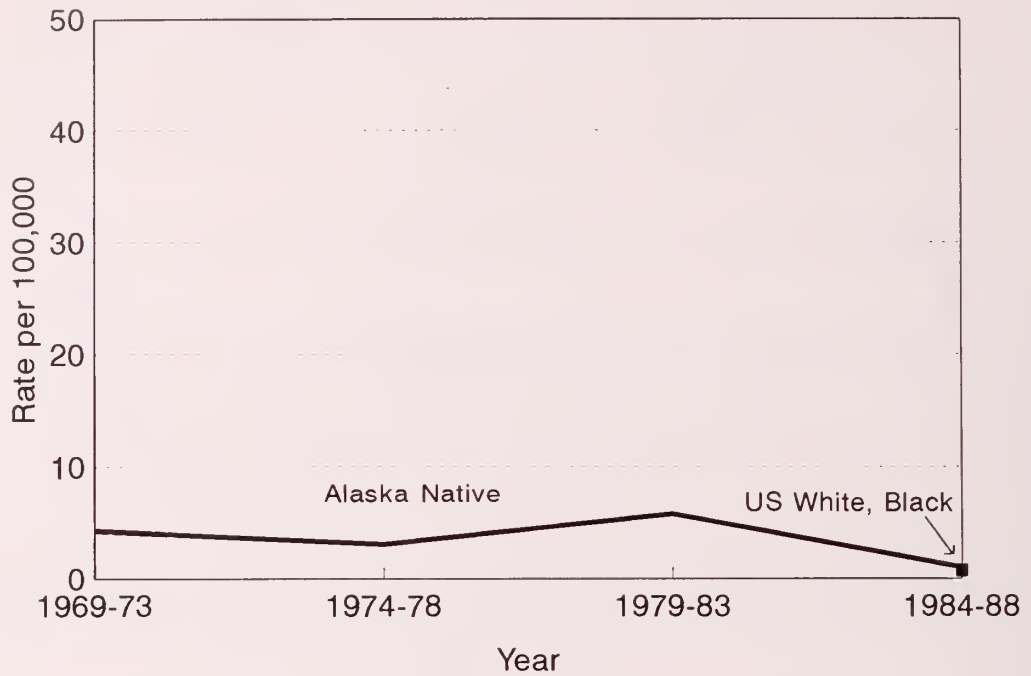
Average Annual Age-Adjusted Cancer Incidence 1969-1988

**Esophagus**  
Rate per 100,000

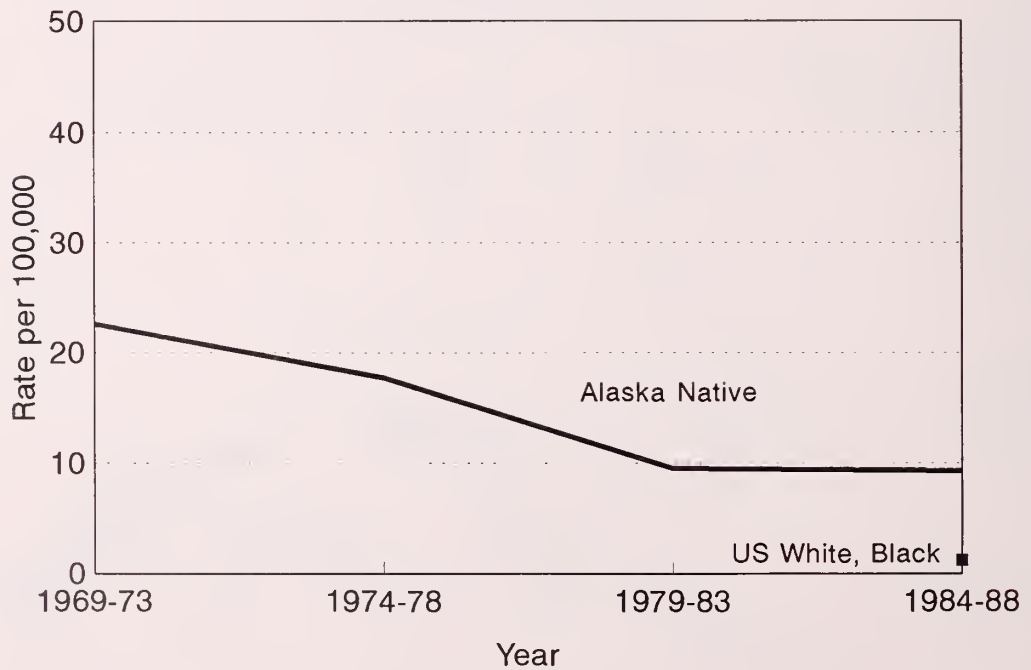


Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988

**Gallbladder: Male**



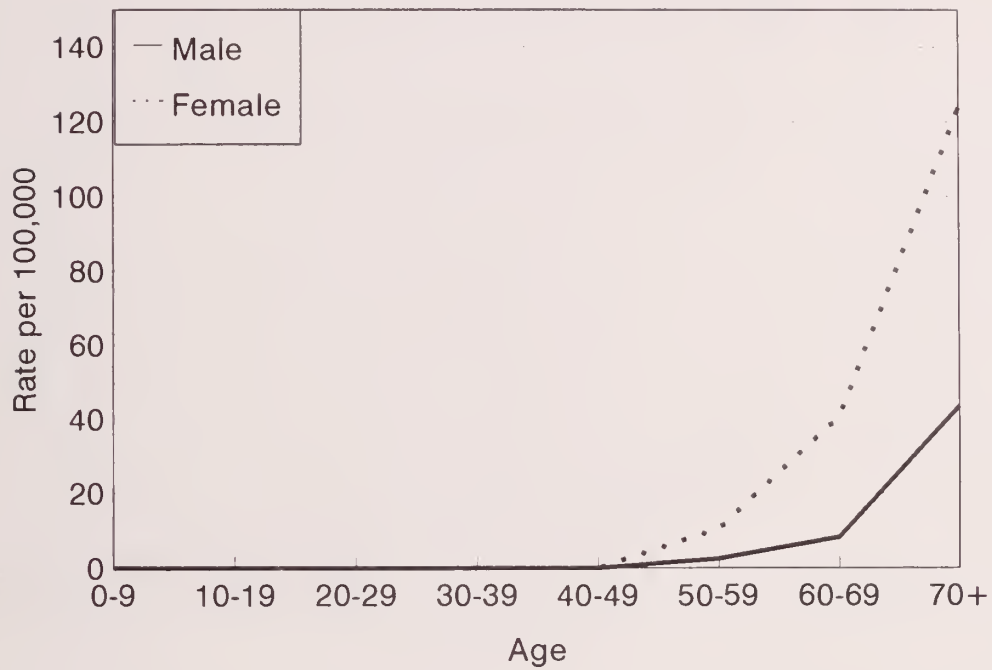
**Gallbladder: Female**



US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.

Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

**Gallbladder**

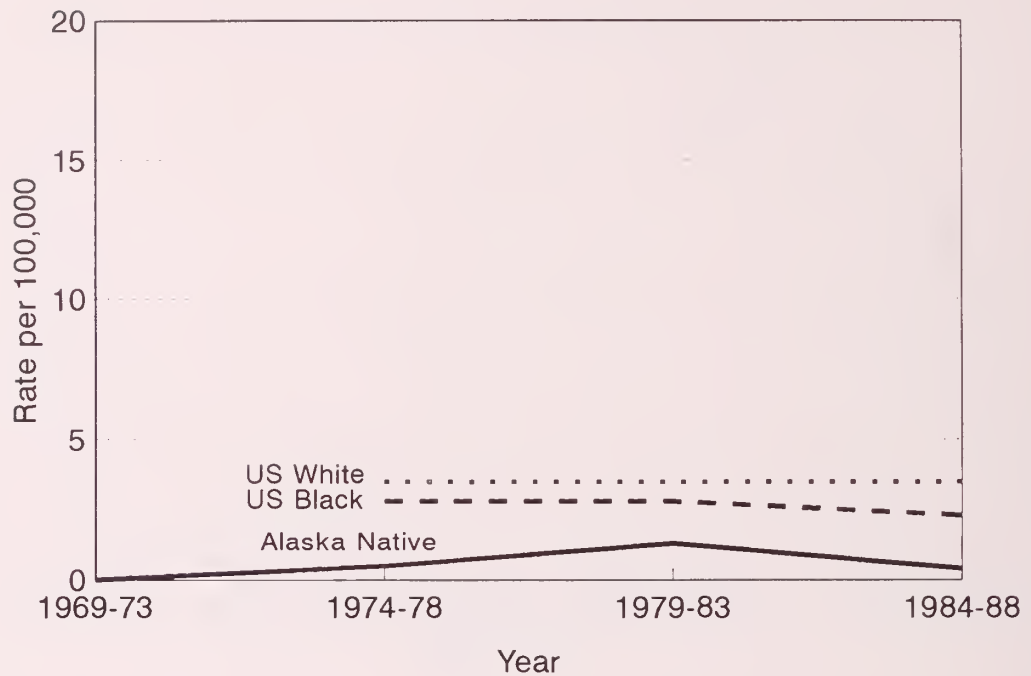


Too few cases to show rates by Service Unit.

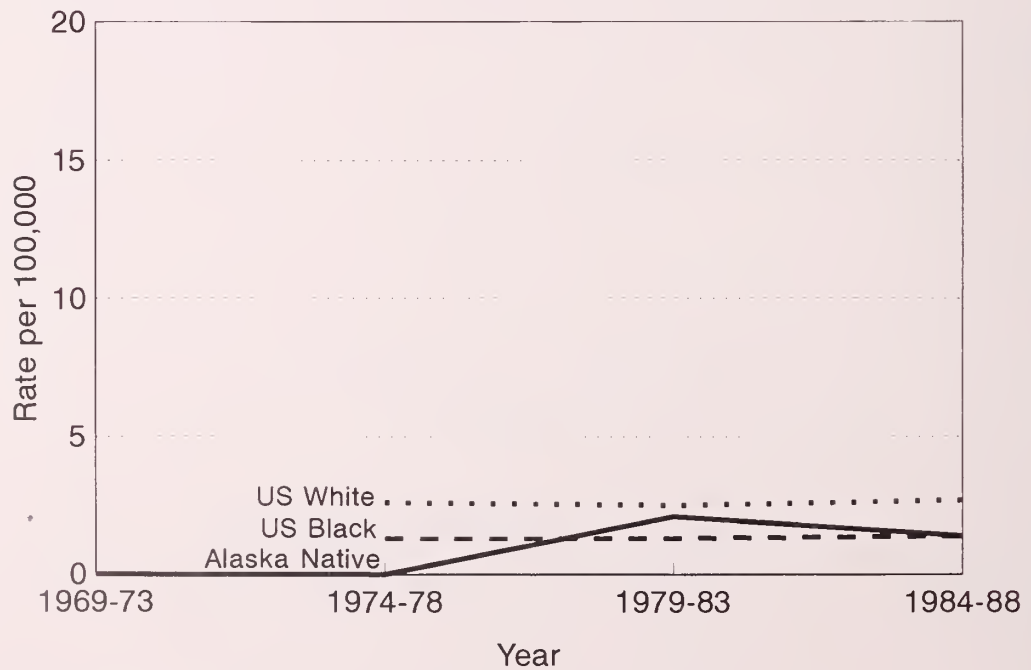


Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US 1974-1988

***Hodgkin's Disease: Male***



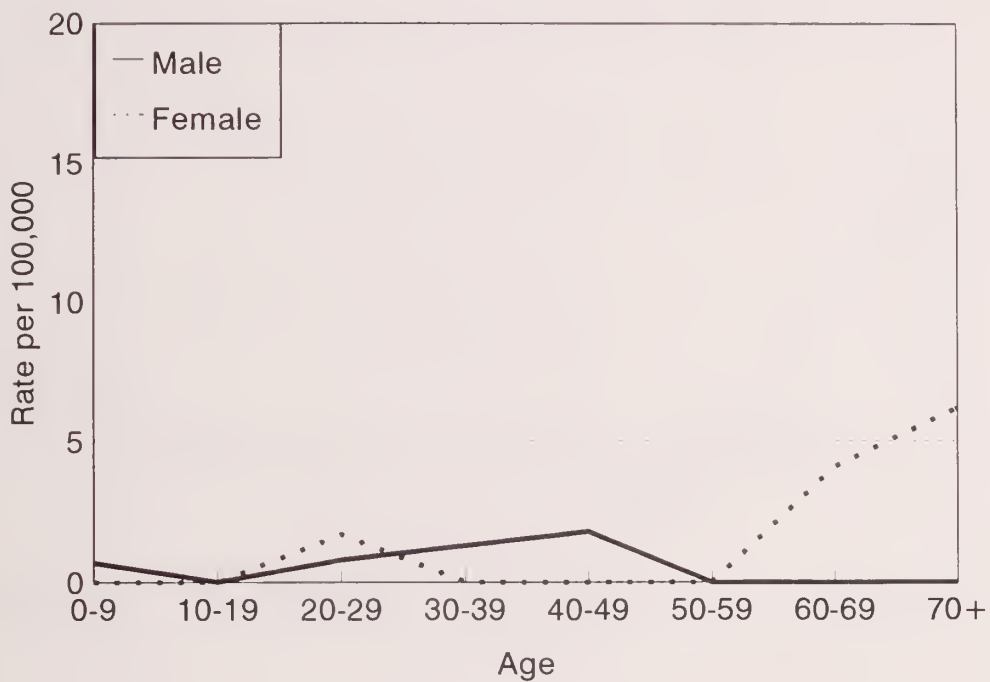
***Hodgkin's Disease: Female***



US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.

Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

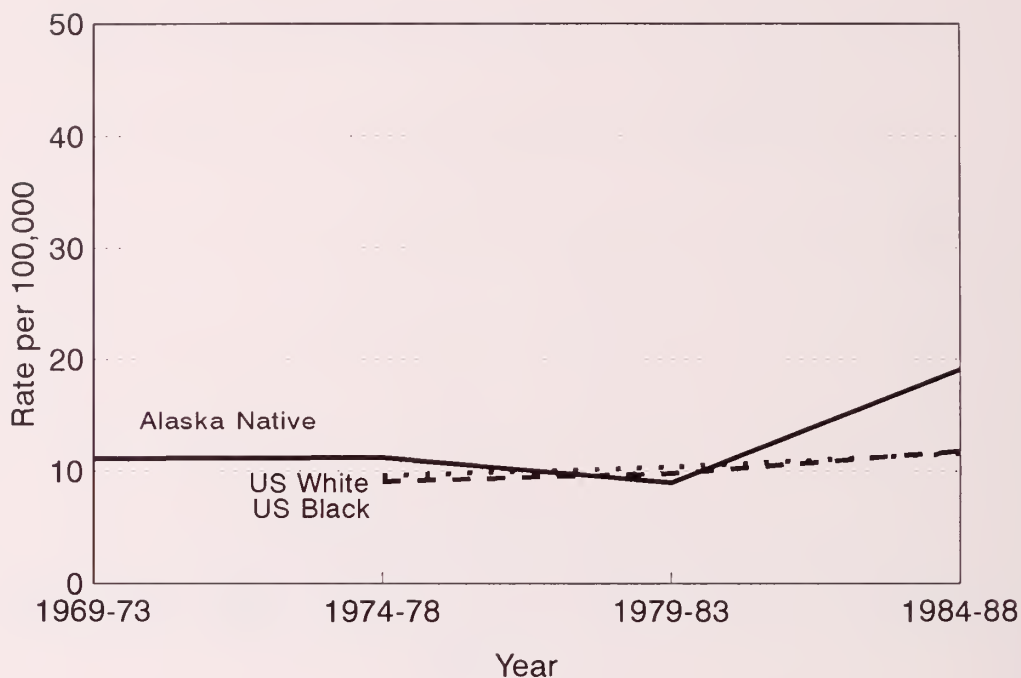
***Hodgkin's Disease***



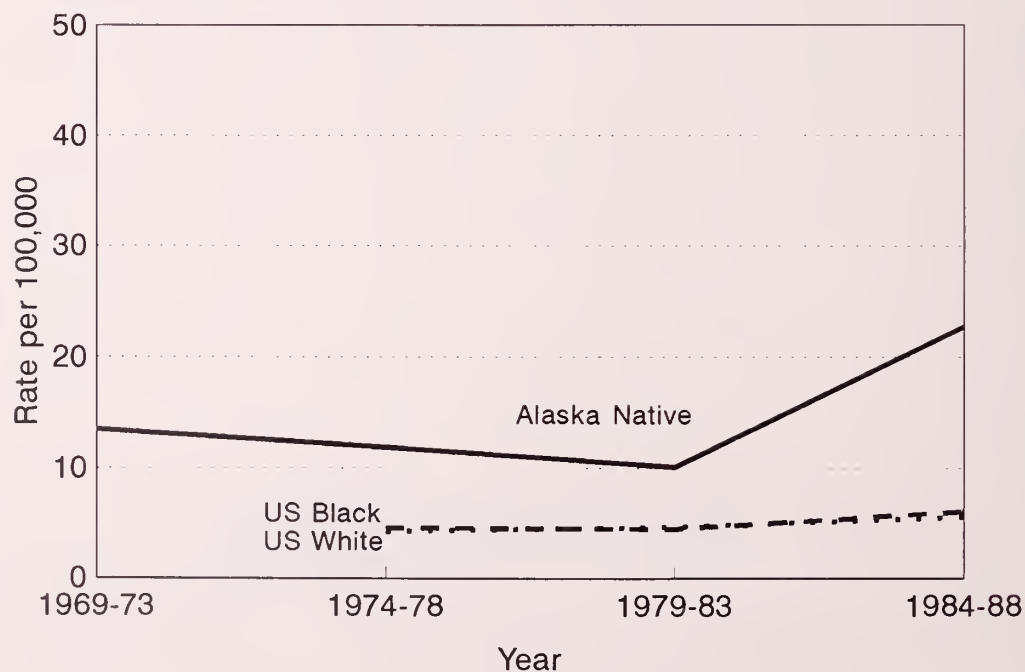
Too few cases to show rates by Service Unit.

Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US 1974-1988

***Kidney and Renal Pelvis: Male***



***Kidney and Renal Pelvis: Female***

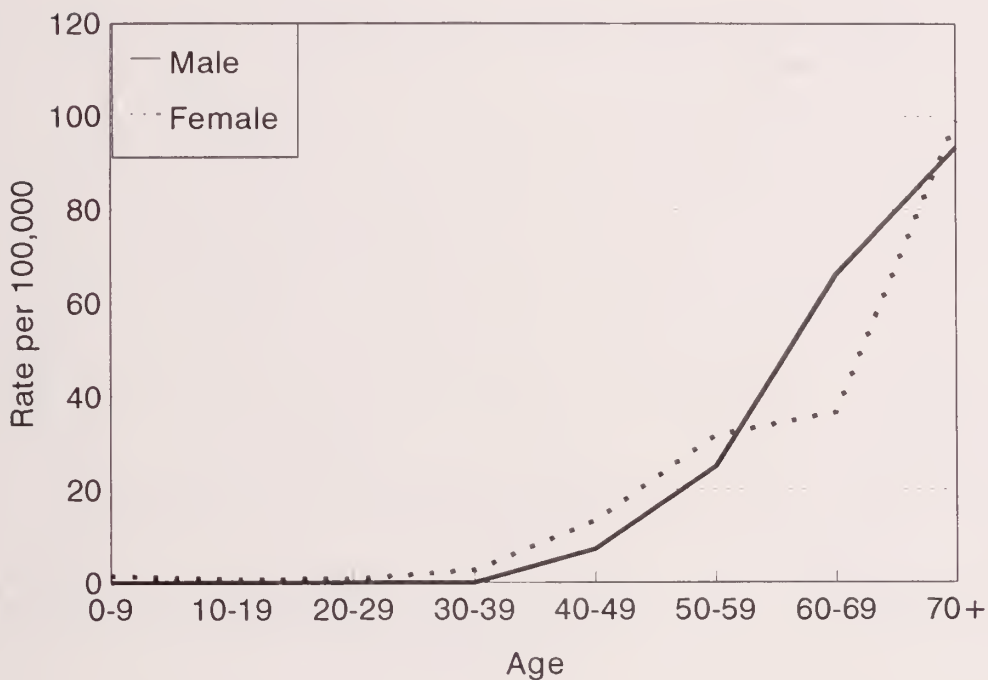


<sup>1</sup>US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.



Average Annual Age Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

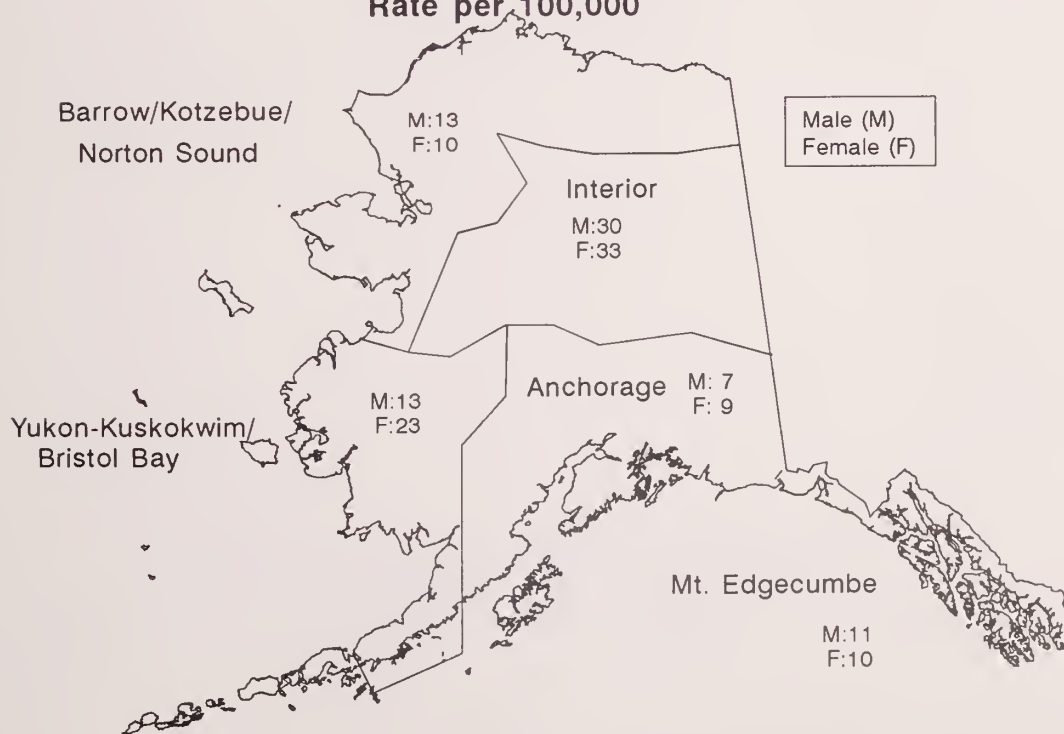
**Kidney and Renal Pelvis**



Average Annual Age-Adjusted Cancer Incidence 1969-1988

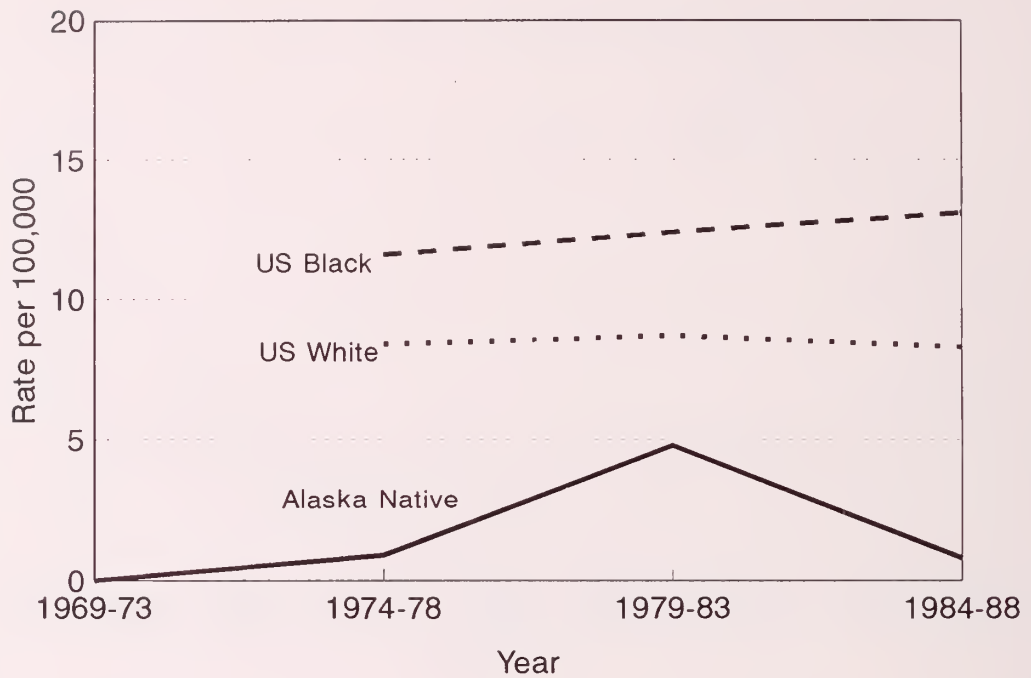
**Kidney and Renal Pelvis**

Rate per 100,000

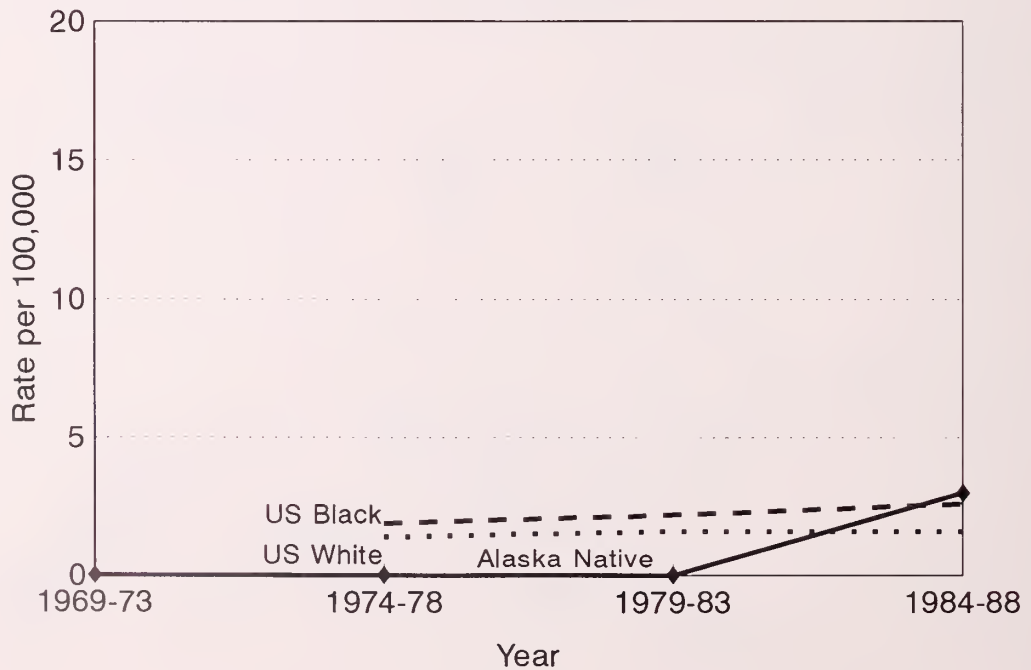


Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US 1974-1988

***Larynx: Male***



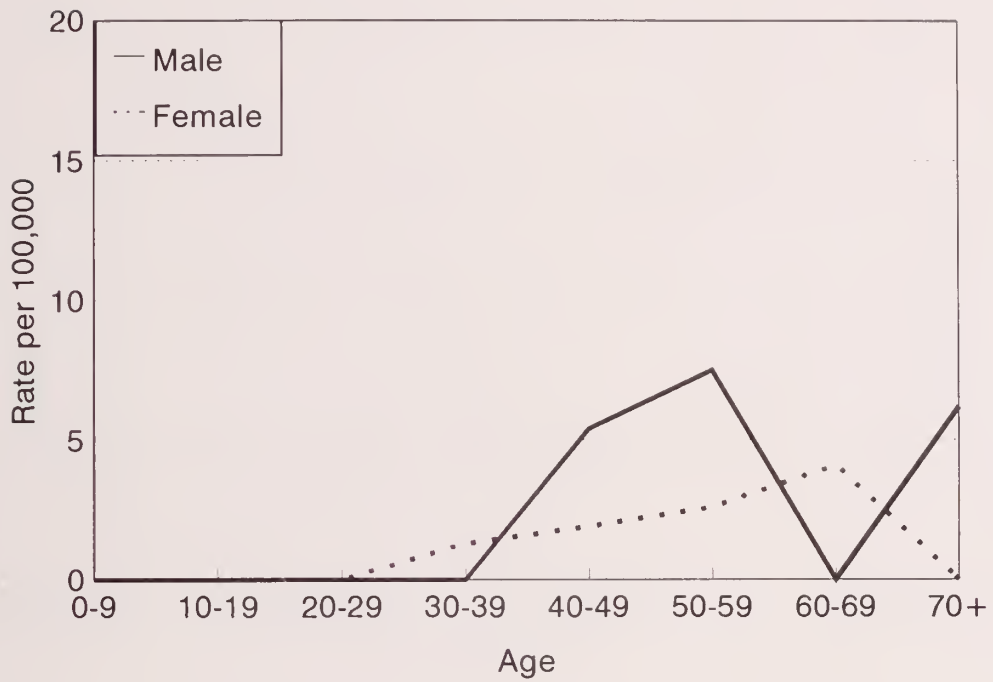
***Larynx: Female***



<sup>1</sup>US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.

Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

**Larynx**

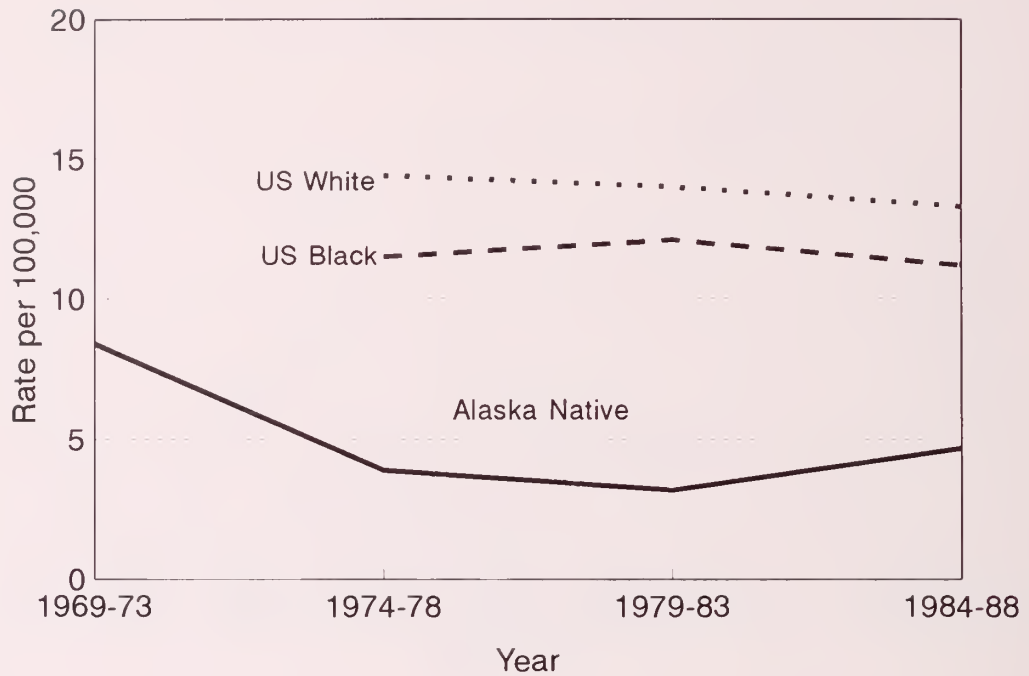


Too few cases to show rates by Service Unit.

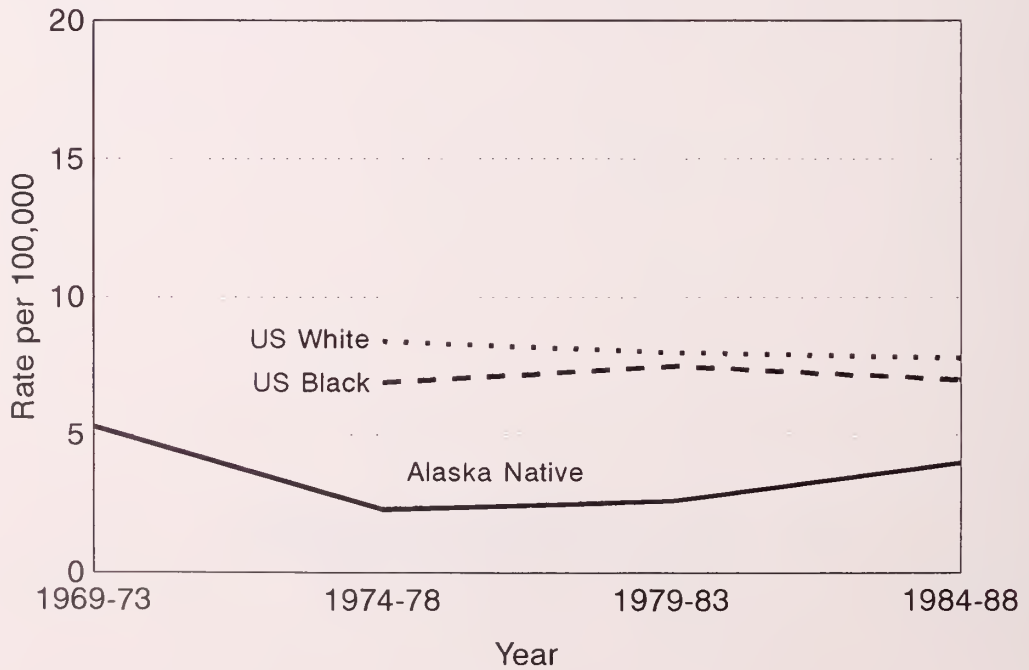


Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US 1974-1988

***Leukemia: Male***



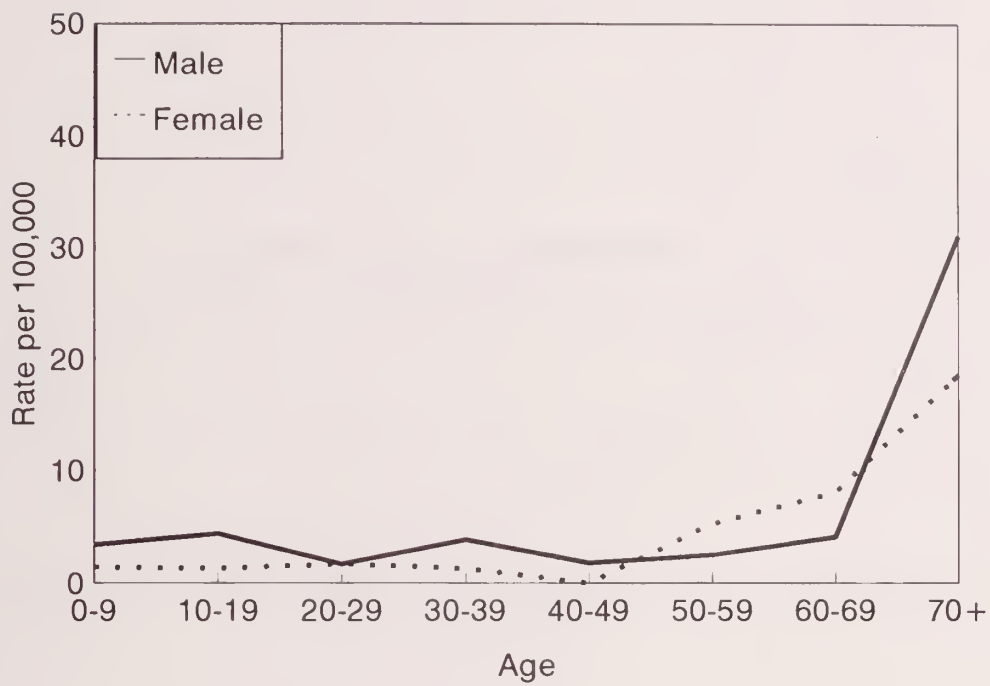
***Leukemia: Female***



<sup>1</sup>US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.

Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

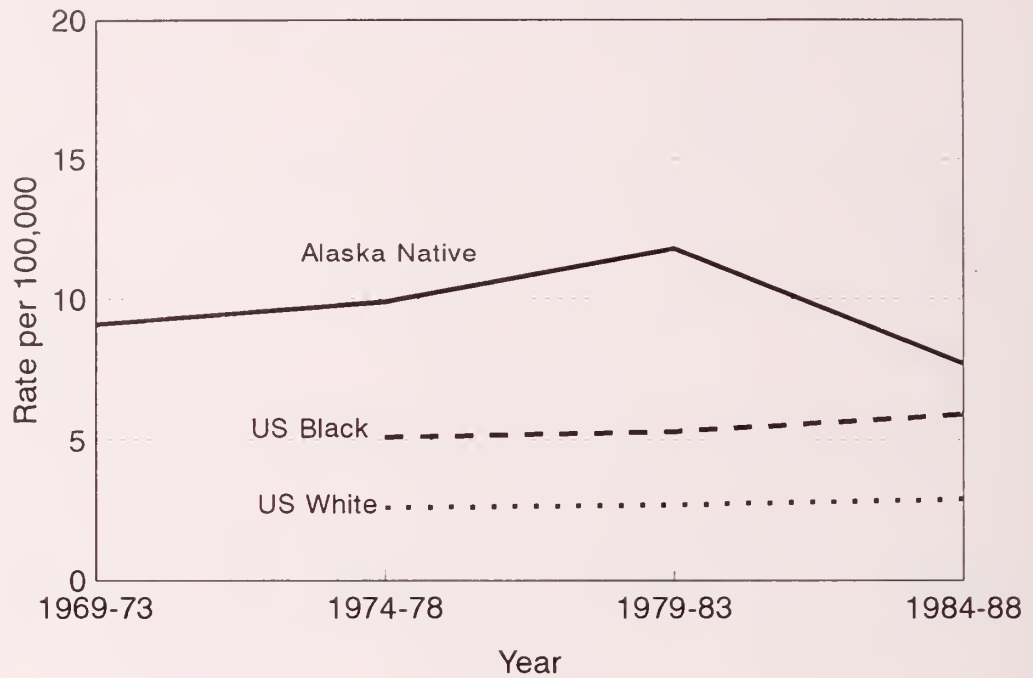
**Leukemia**



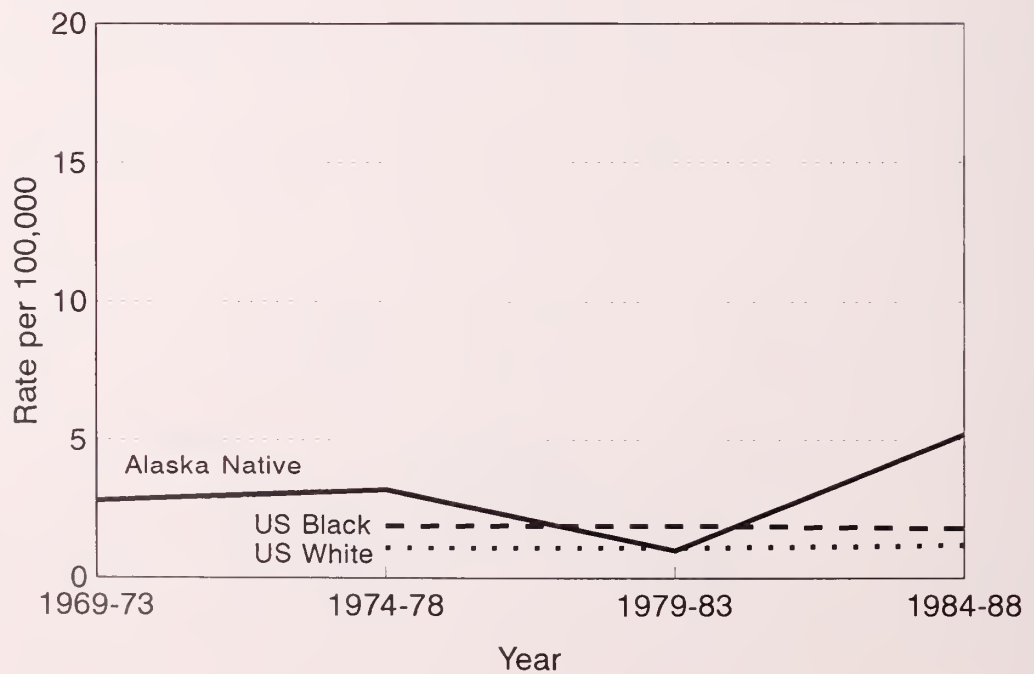
Too few cases to show rates by Service Unit.

Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US 1974-1988

***Liver: Male***



***Liver: Female***

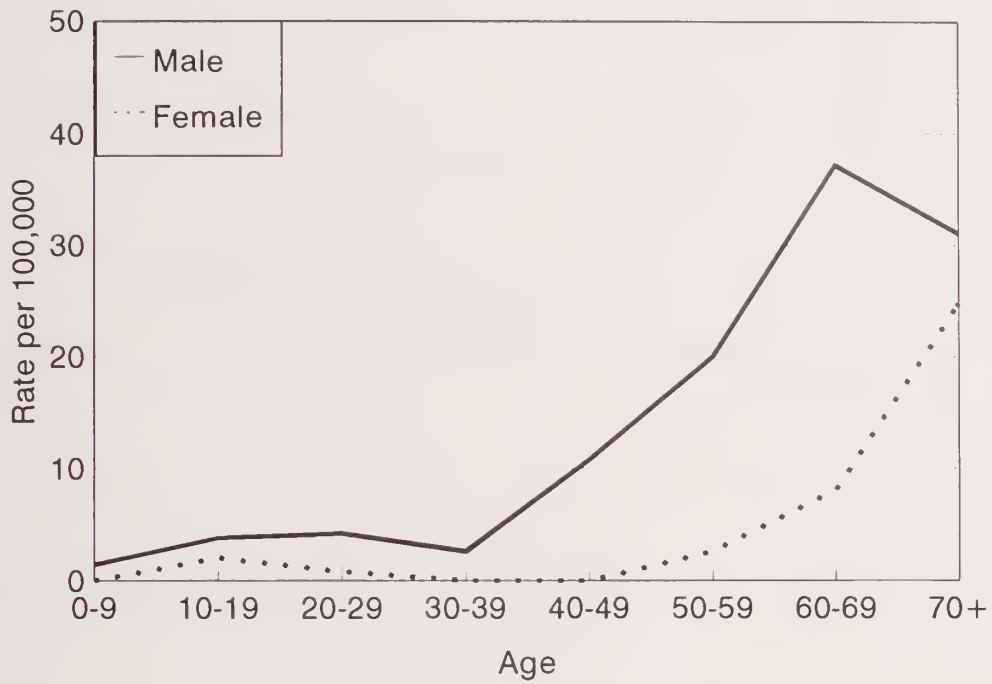


<sup>1</sup>US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.



Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

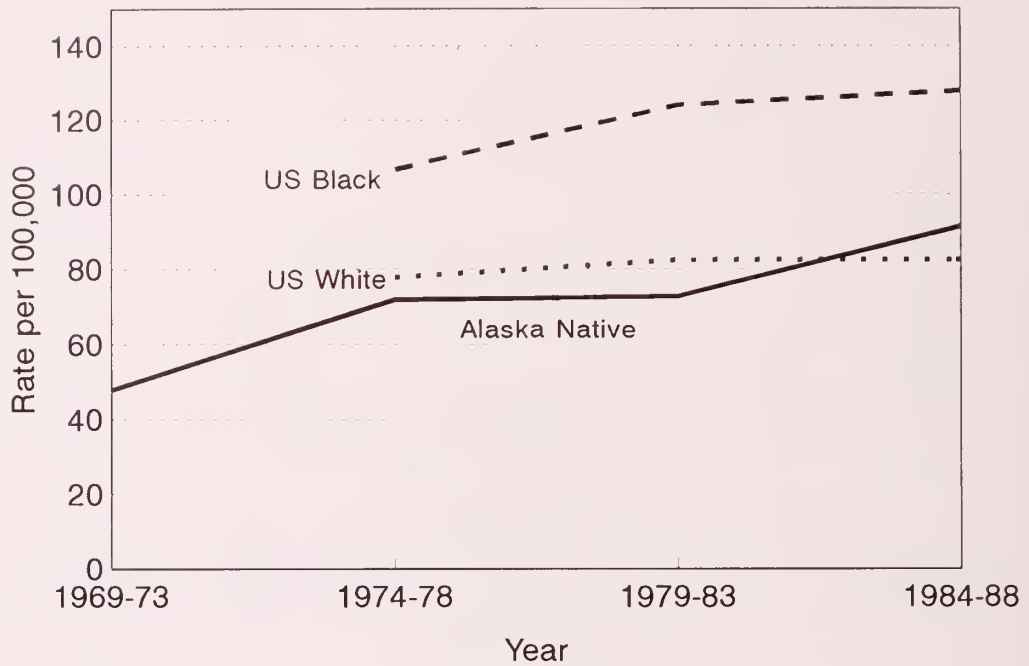
**Liver**



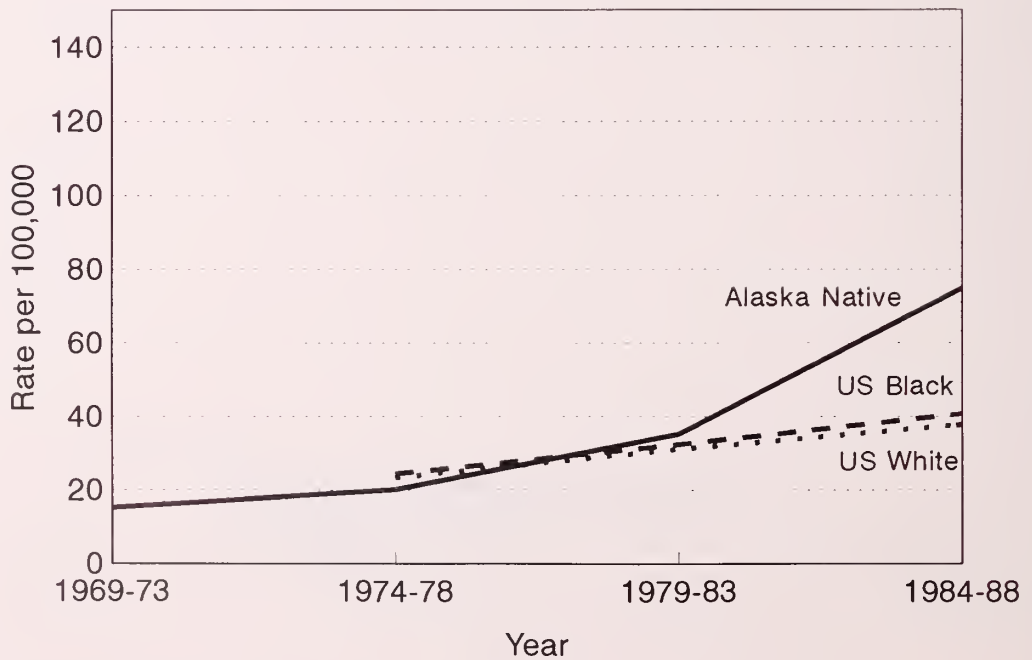
Too few cases to show rates by Service Unit.

Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US 1974-1988

***Lung and Bronchus: Male***

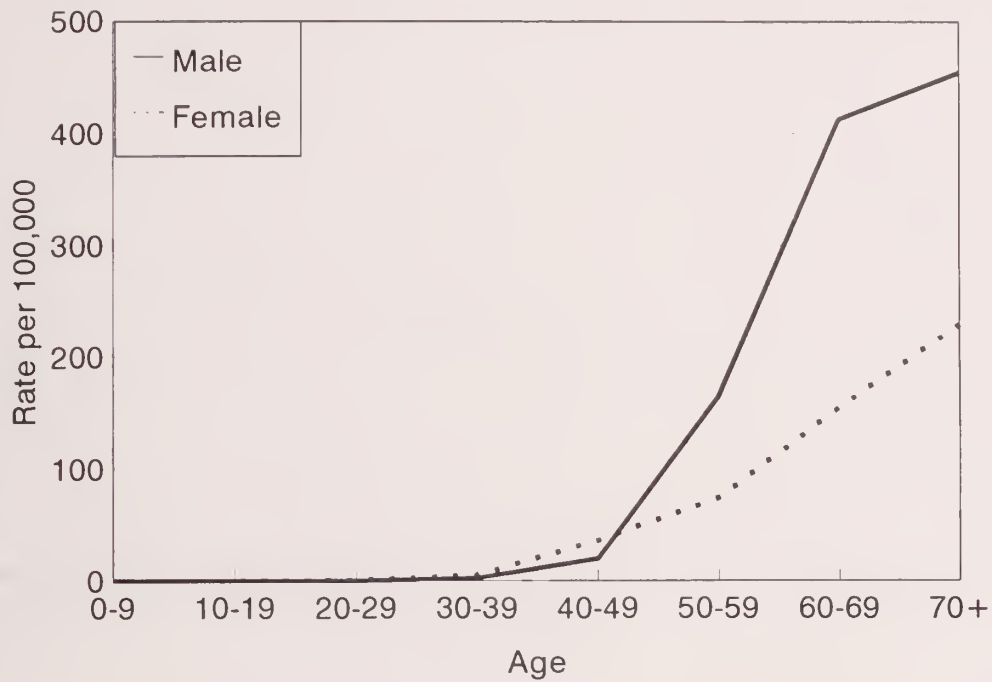


***Lung and Bronchus: Female***

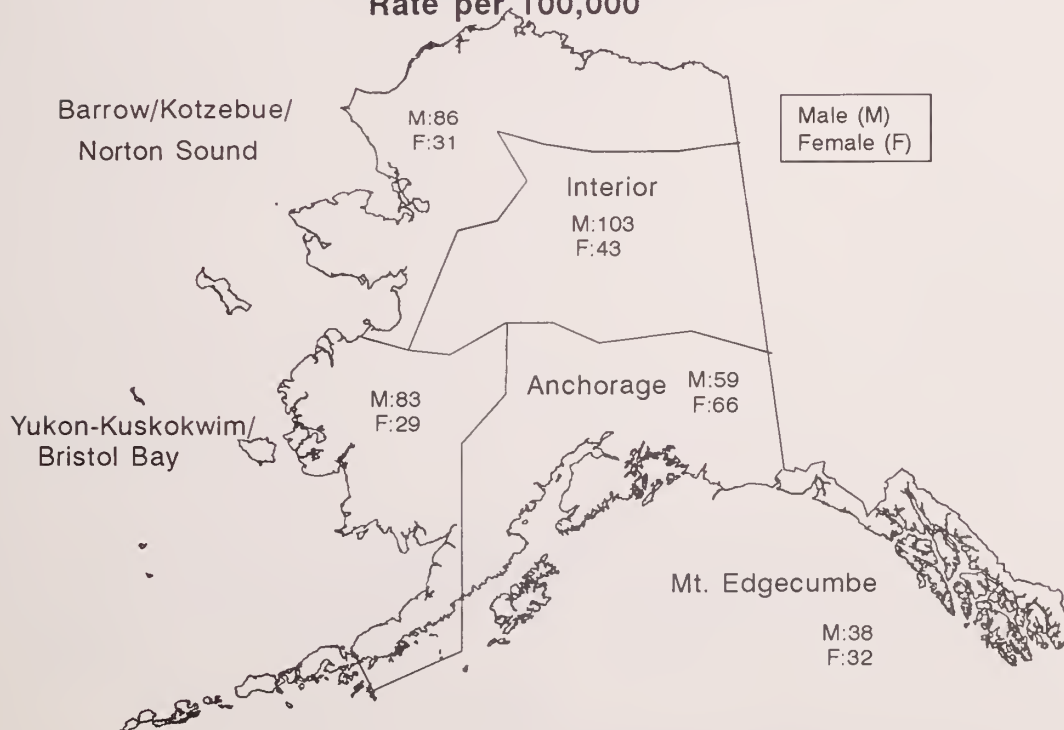


US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.

Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988  
**Lung and Bronchus**



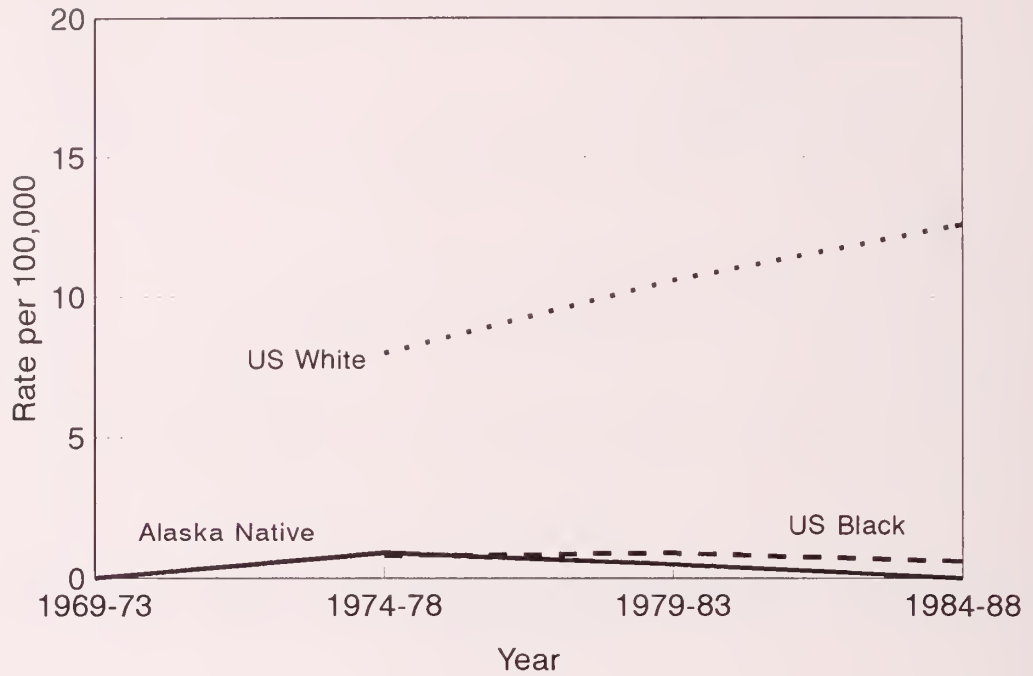
Average Annual Age-Adjusted Cancer Incidence 1969-1988  
**Lung and Bronchus**  
Rate per 100,000



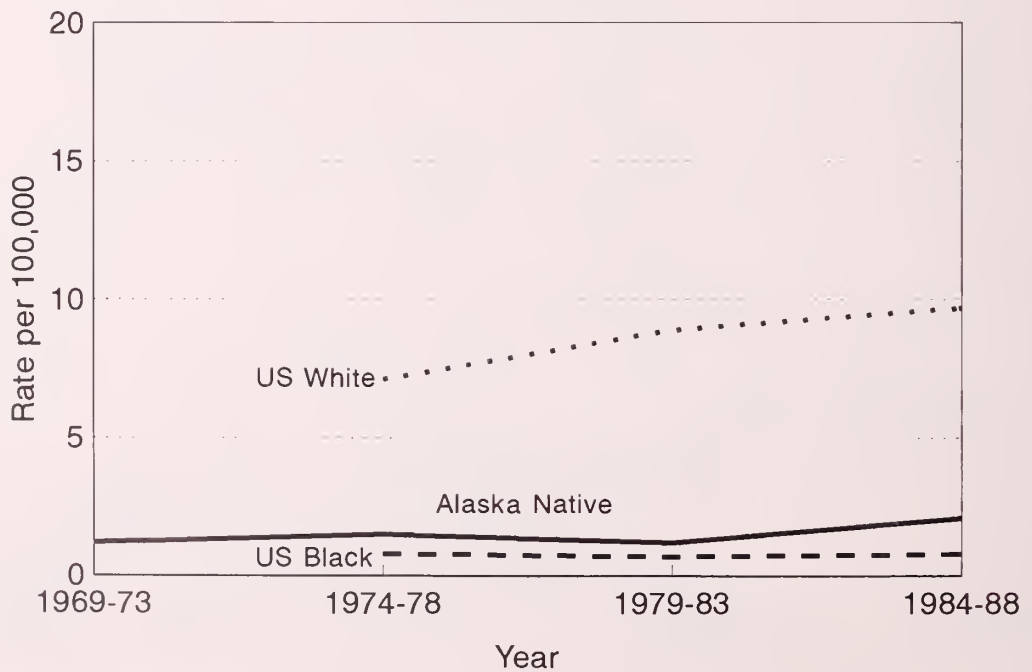


Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US 1974-1988

***Melanoma of Skin: Male***



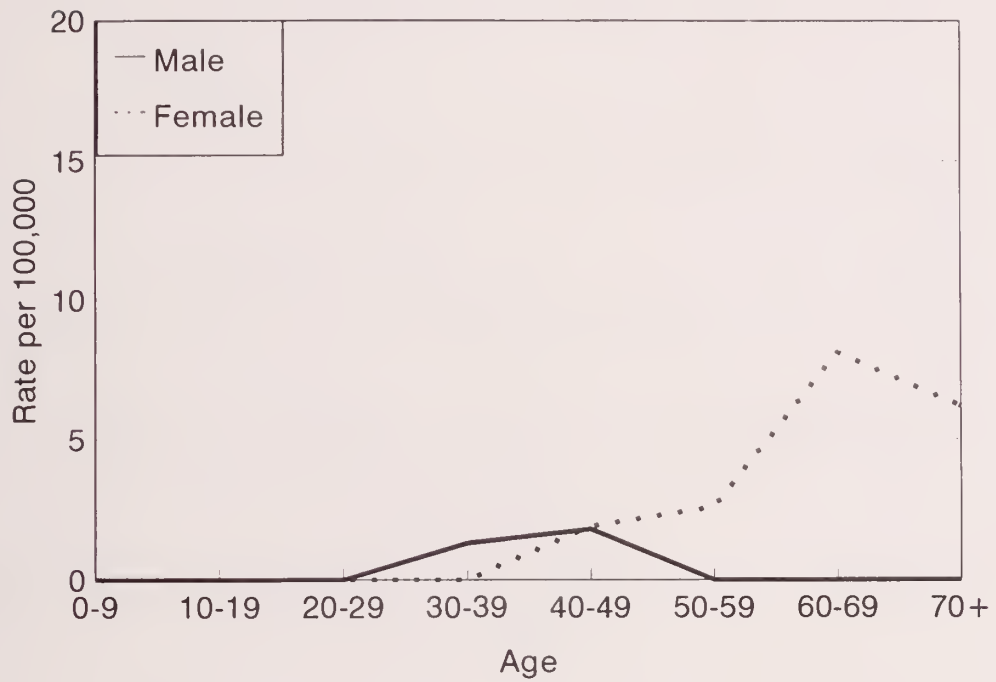
***Melanoma of Skin: Female***



<sup>1</sup>US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.

Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

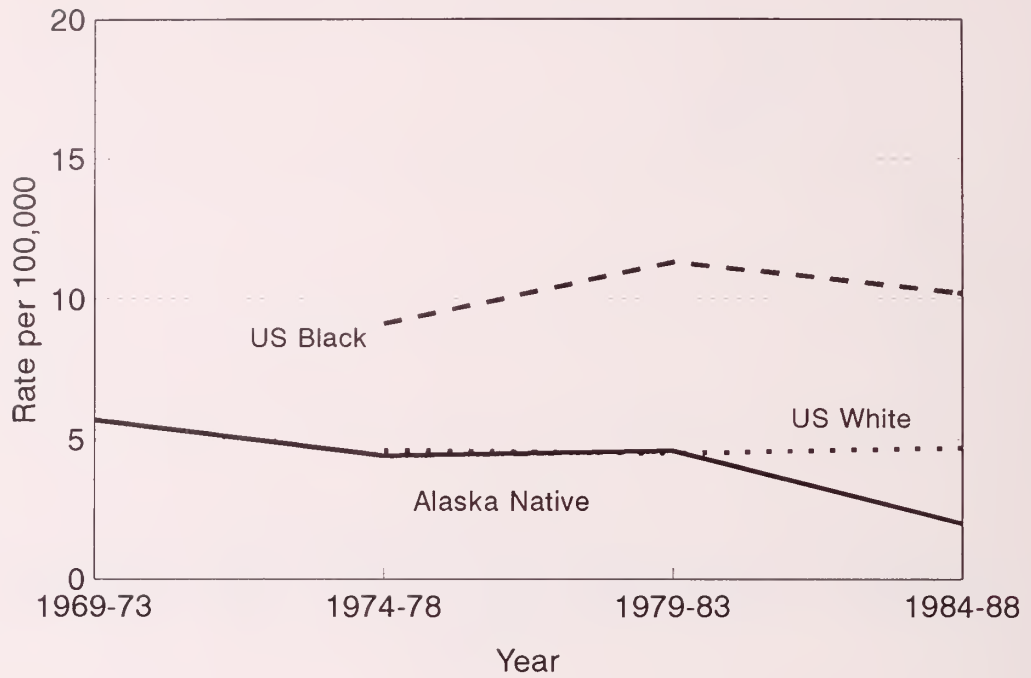
***Melanoma of Skin***



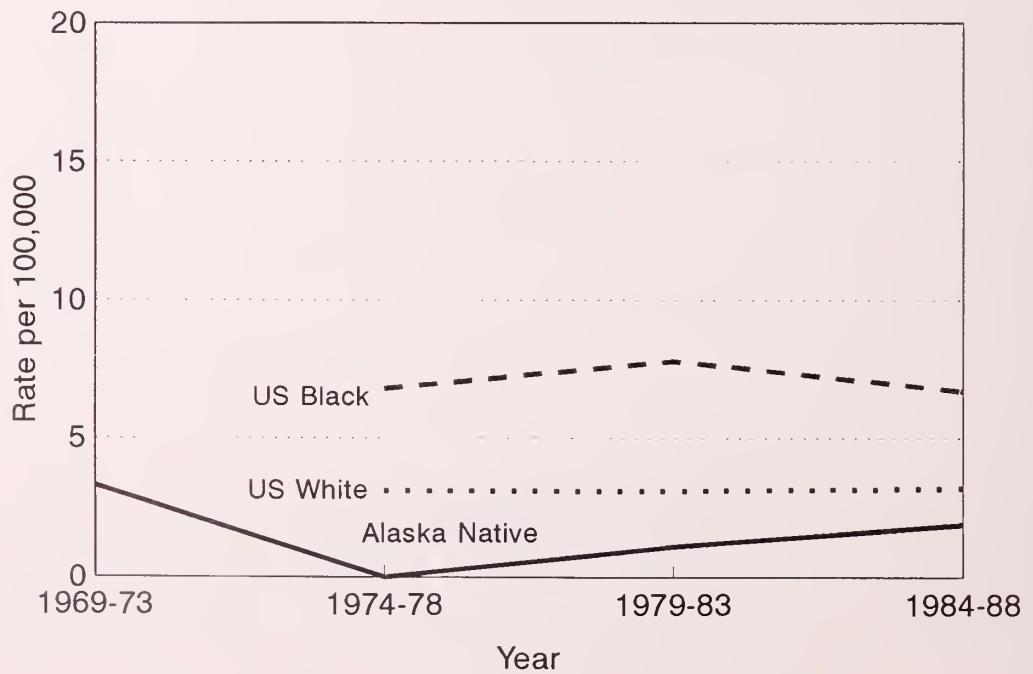
Too few cases to show rates by Service Unit.

Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US 1974-1988

***Multiple Myeloma: Male***



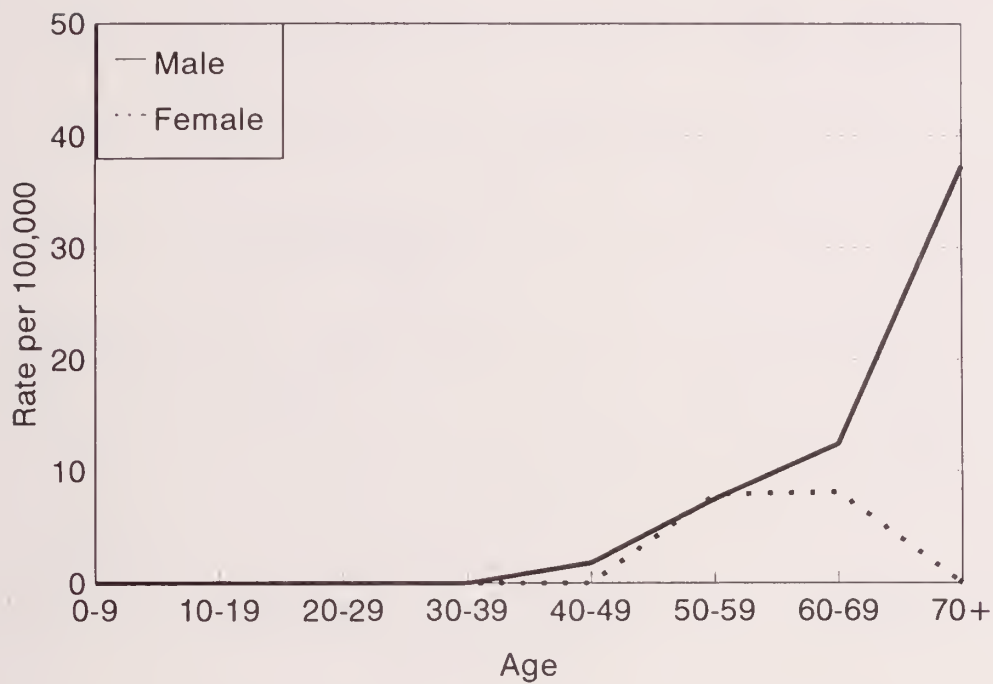
***Multiple Myeloma: Female***



<sup>1</sup>US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.



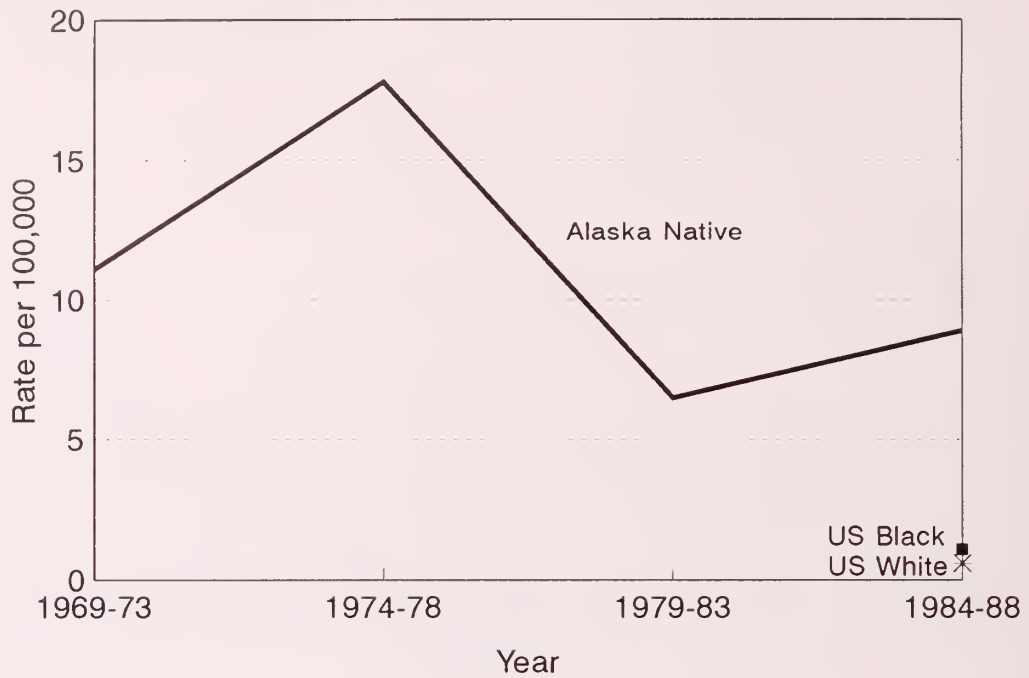
Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988  
**Multiple Myeloma**



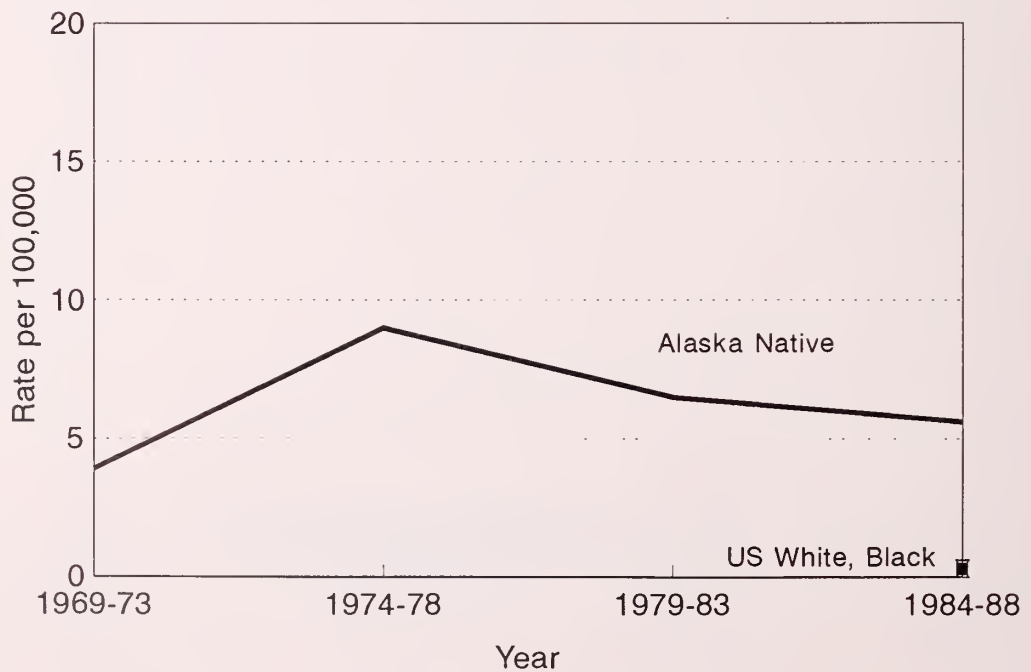
Too few cases to show rates by Service Unit.

Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988

***Nasopharynx: Male***



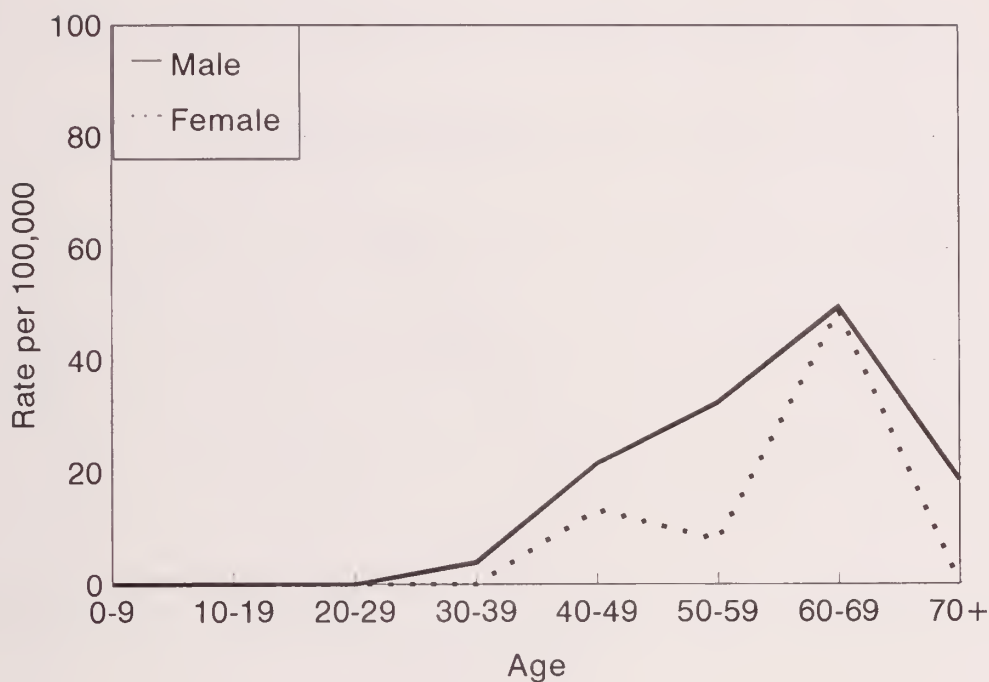
***Nasopharynx: Female***



<sup>1</sup>US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.

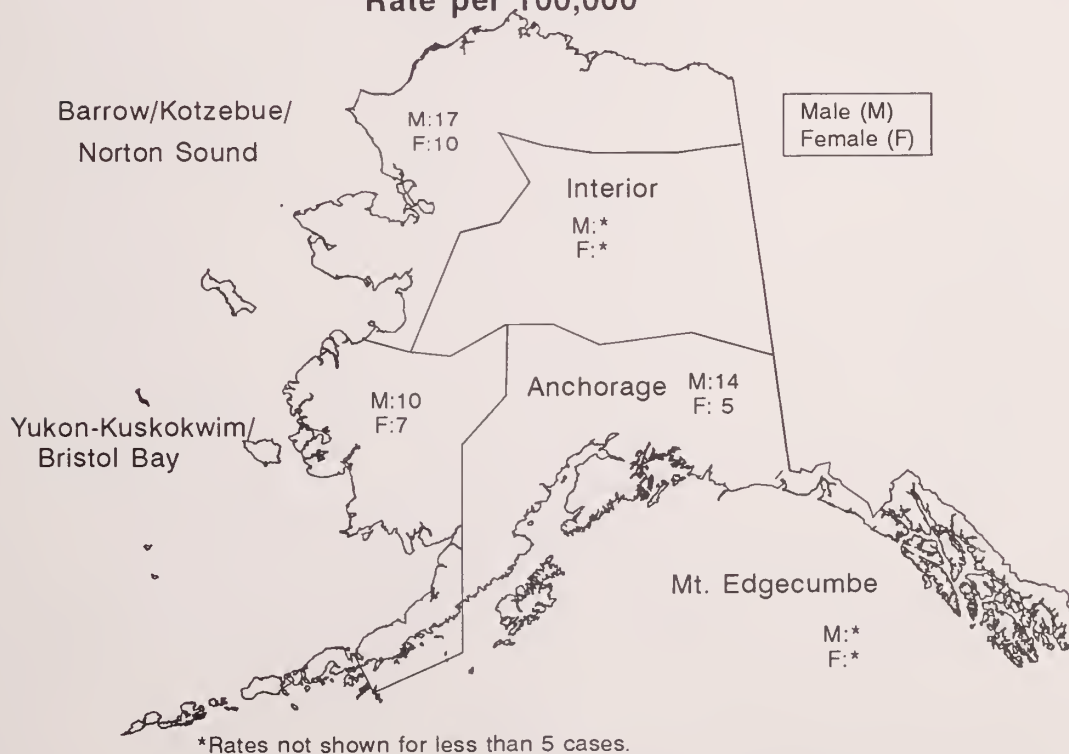
Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

**Nasopharynx**



Average Annual Age-Adjusted Cancer Incidence 1969-1988

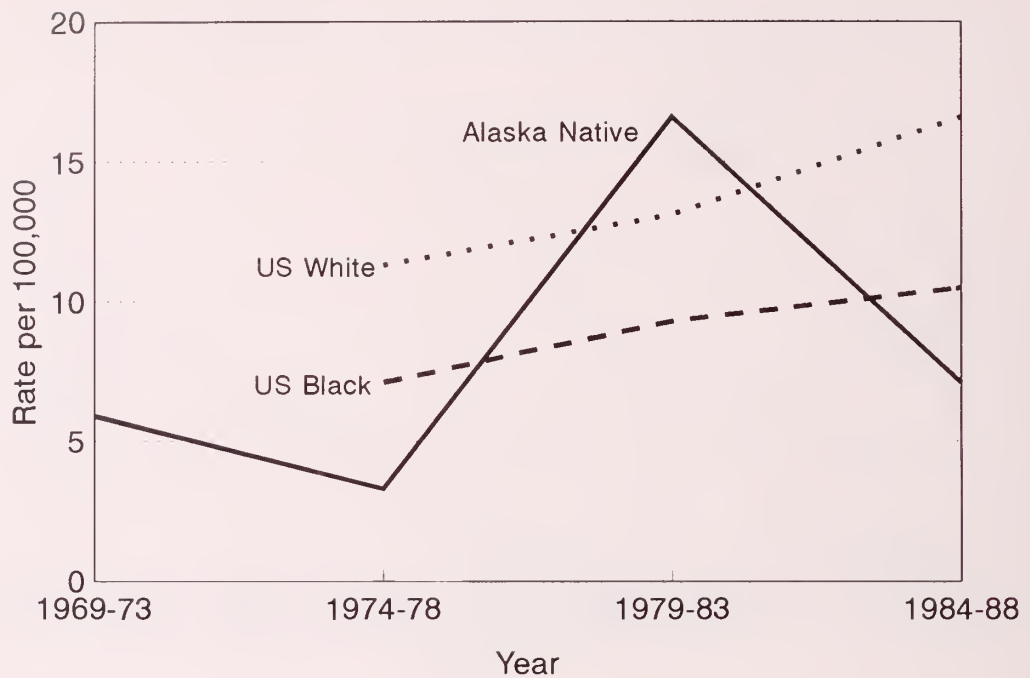
**Nasopharynx**  
Rate per 100,000



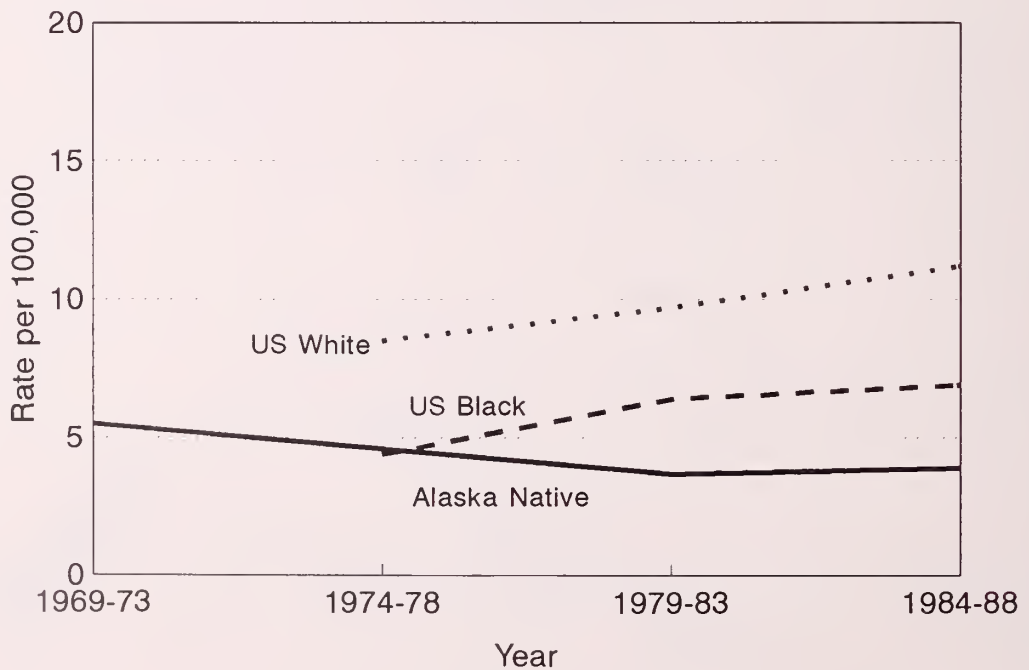


Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US<sup>1</sup> 1974-1988

***Non-Hodgkin's Lymphoma: Male***



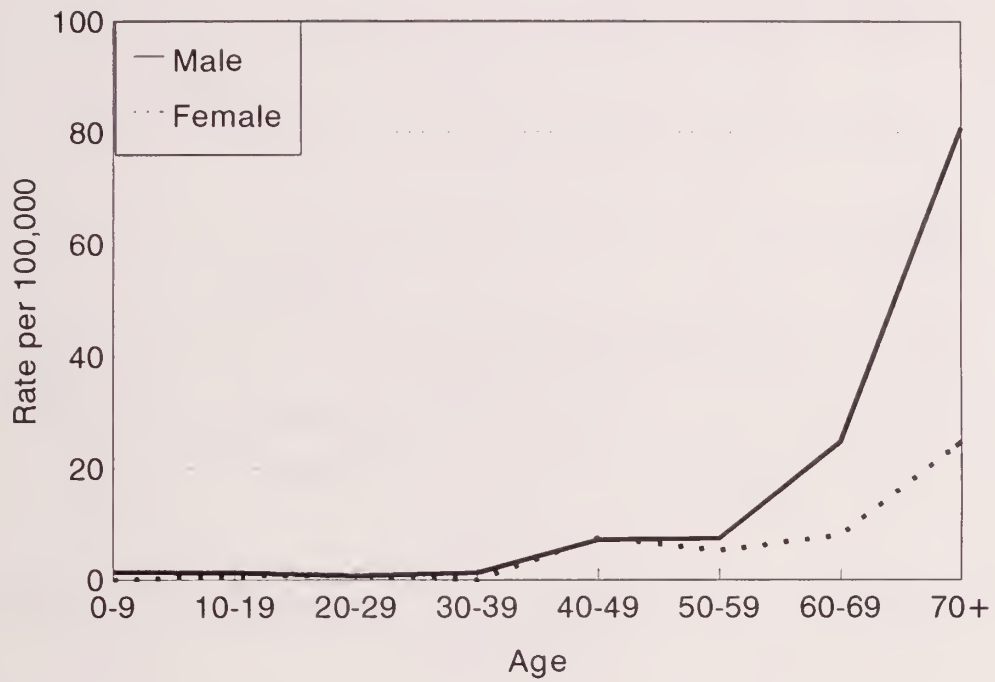
***Non-Hodgkin's Lymphoma: Female***



<sup>1</sup>US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.

Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

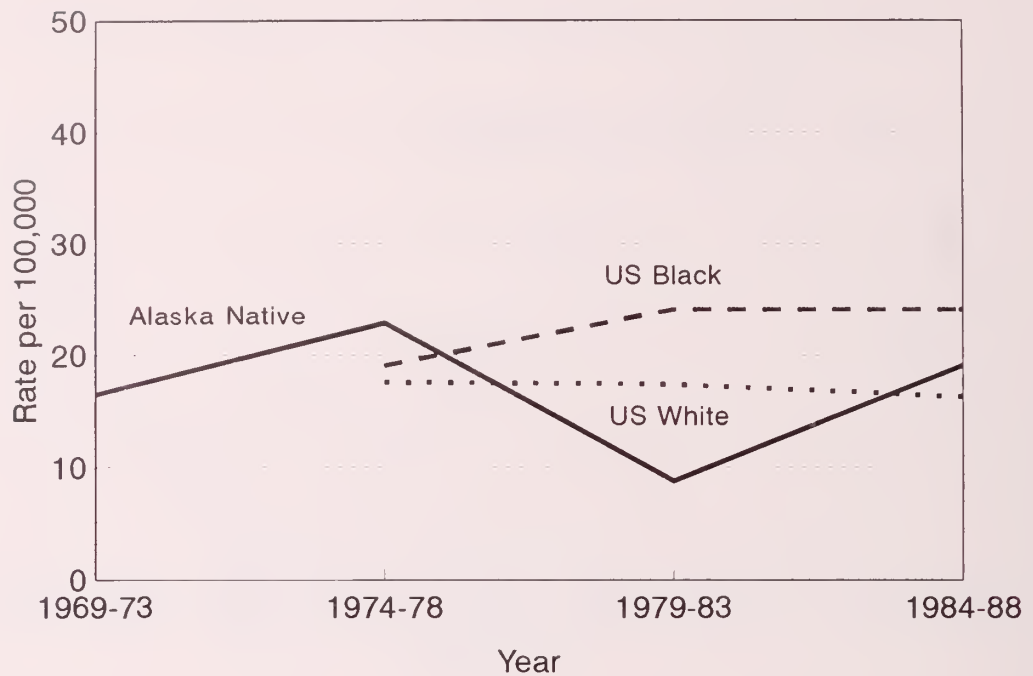
***Non-Hodgkin's Lymphoma***



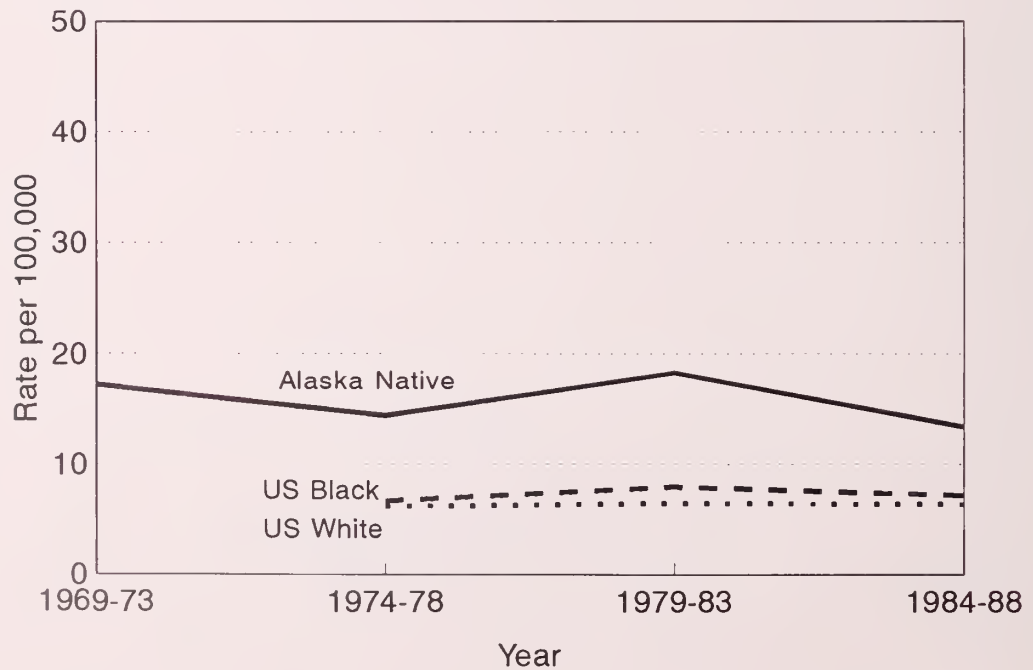
Too few cases to show rates by Service Unit.

Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US<sup>1</sup> 1974-1988

**Oral Cavity and Pharynx: Male**



**Oral Cavity and Pharynx: Female**

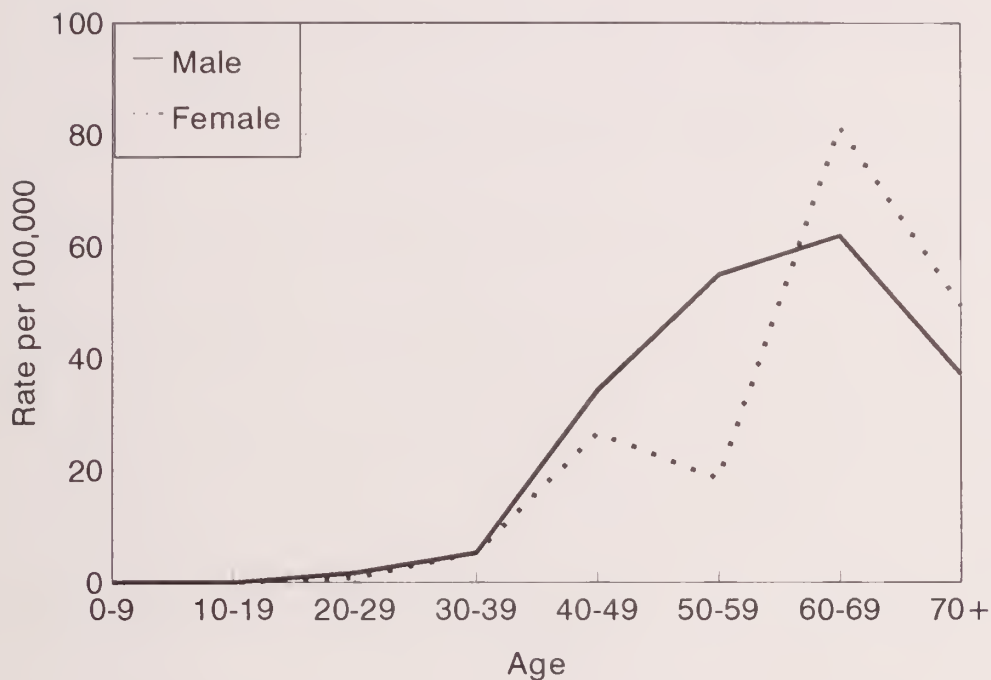


<sup>1</sup>US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.



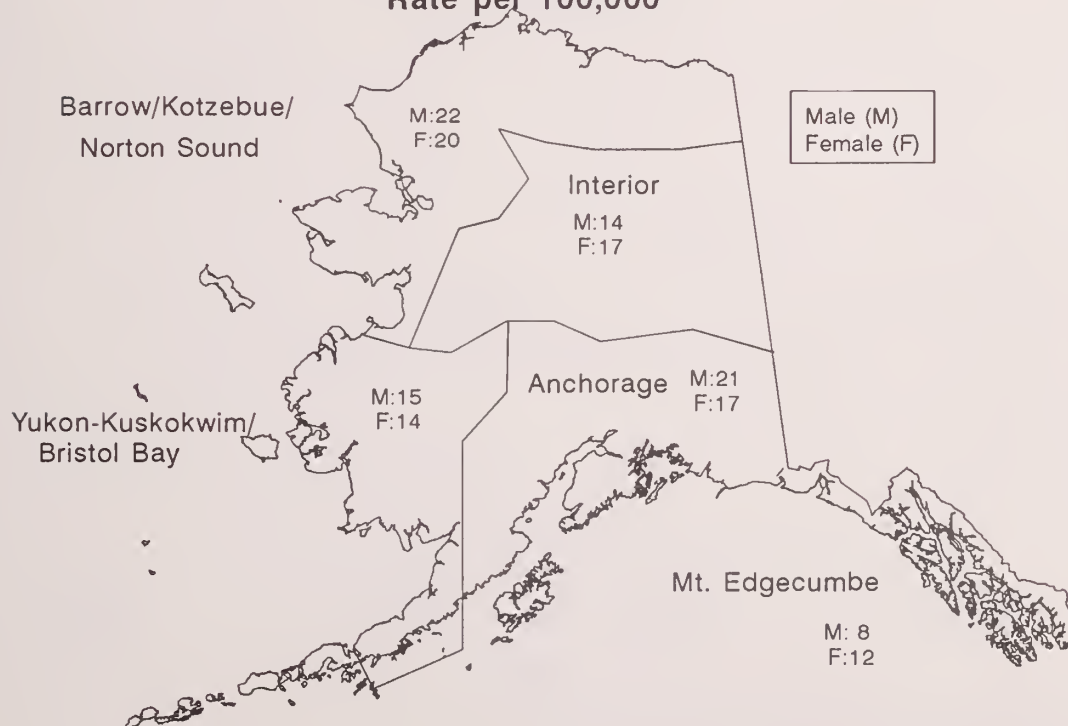
Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

**Oral Cavity and Pharynx**

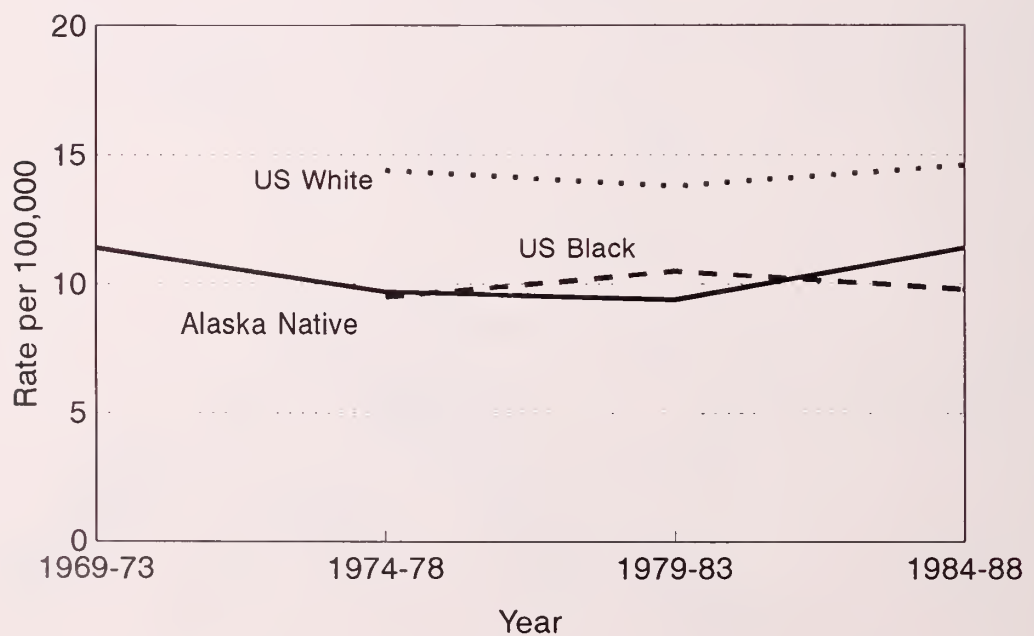


Average Annual Age-Adjusted Cancer Incidence 1969-1988

**Oral Cavity and Pharynx**  
Rate per 100,000



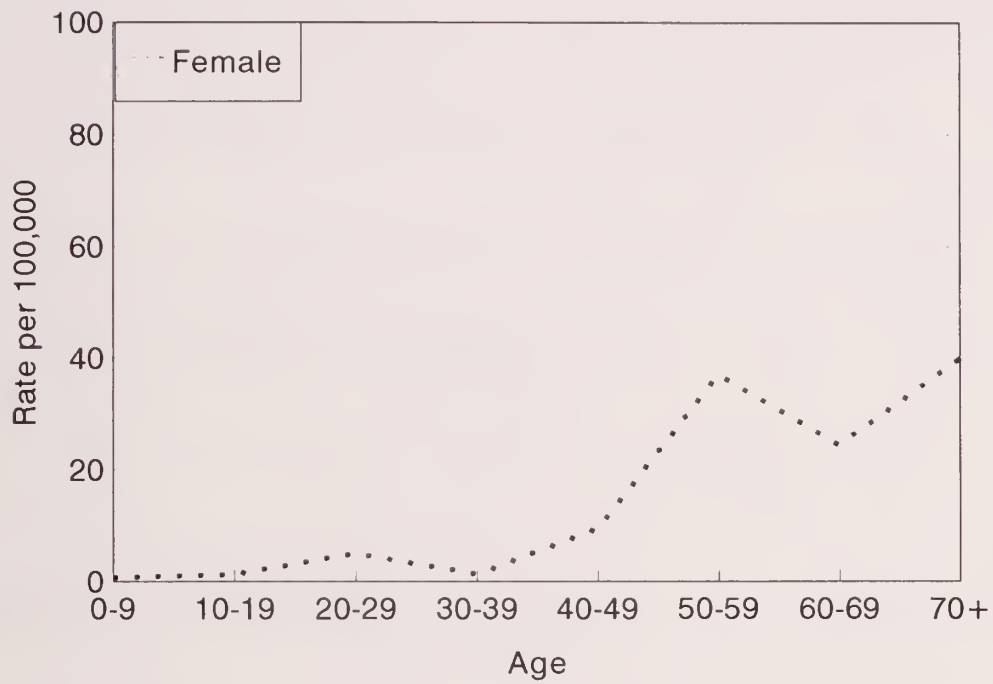
Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US 1974-1988  
**Ovary**



<sup>1</sup>US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.

Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

**Ovary**

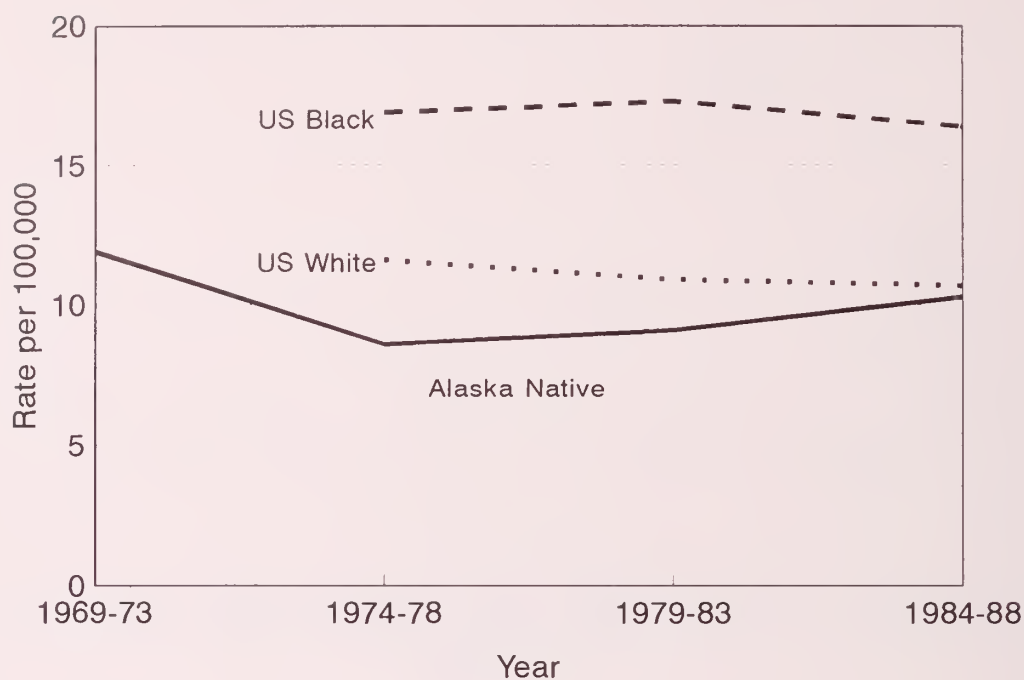


Too few cases to show rates by Service Unit.

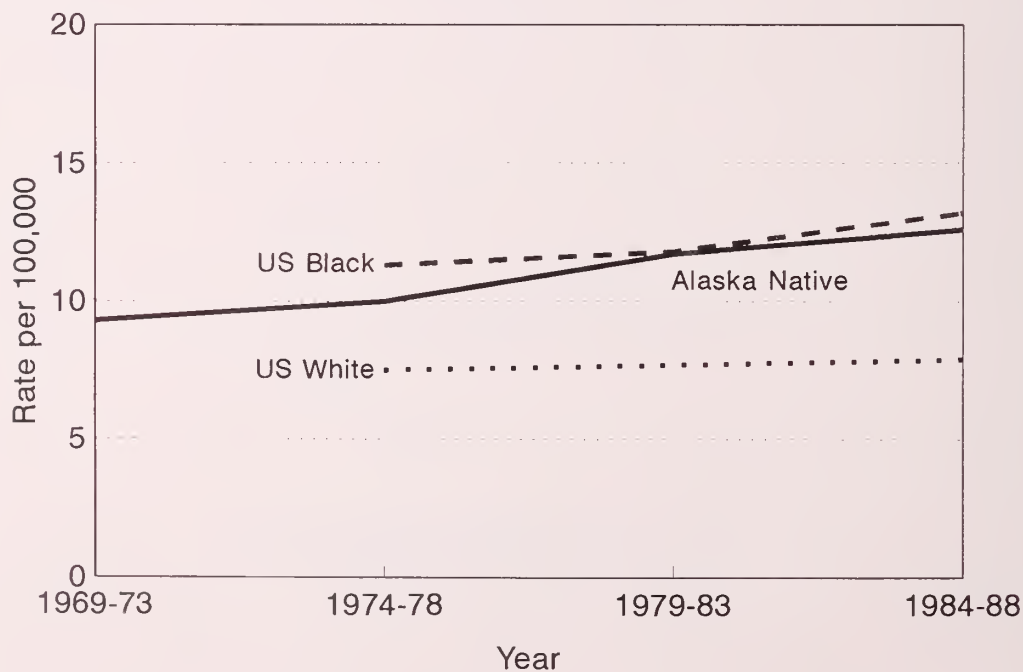


Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US<sup>1</sup> 1974-1988

***Pancreas: Male***



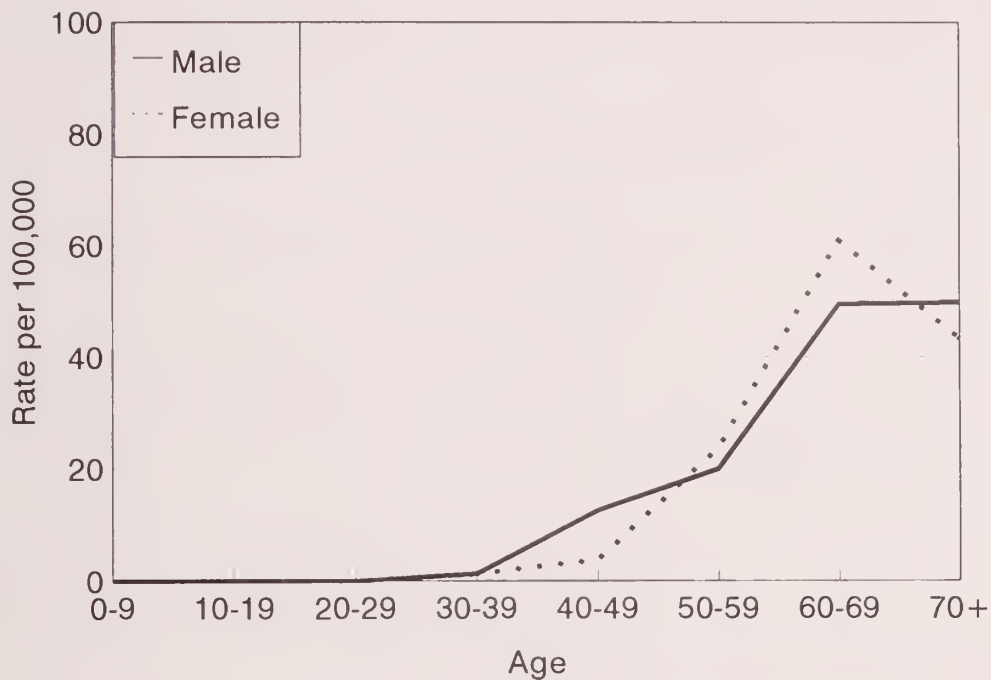
***Pancreas: Female***



<sup>1</sup>US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.

Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

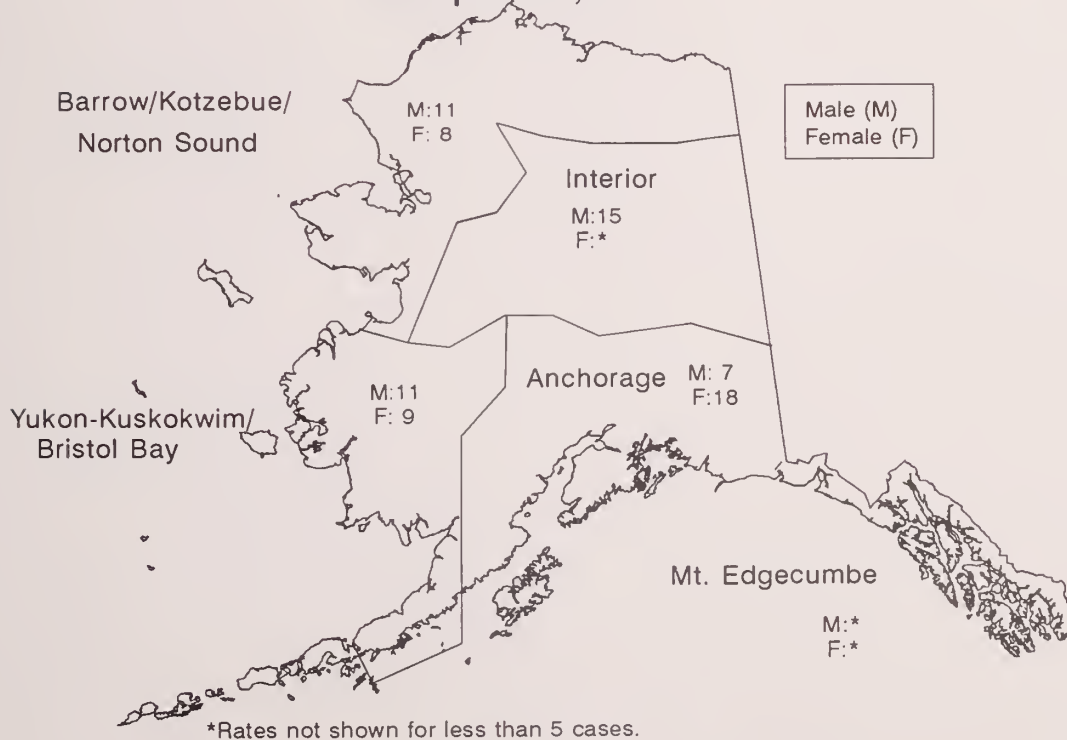
**Pancreas**



Average Annual Age-Adjusted Cancer Incidence 1969-1988

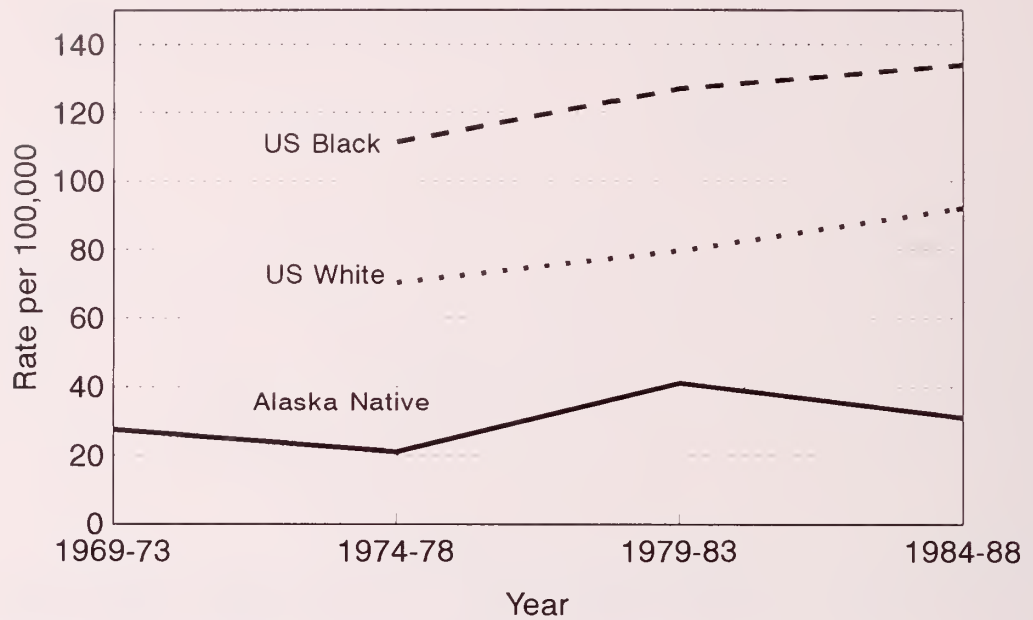
**Pancreas**

Rate per 100,000



Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988, US 1974-1988

**Prostate**

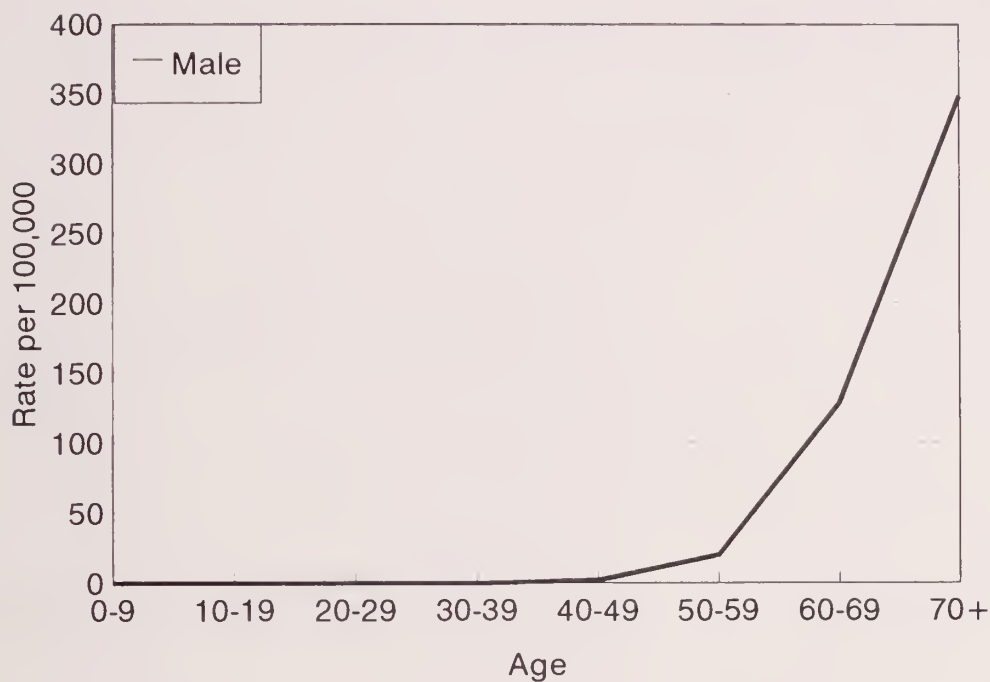


<sup>1</sup>US Rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.



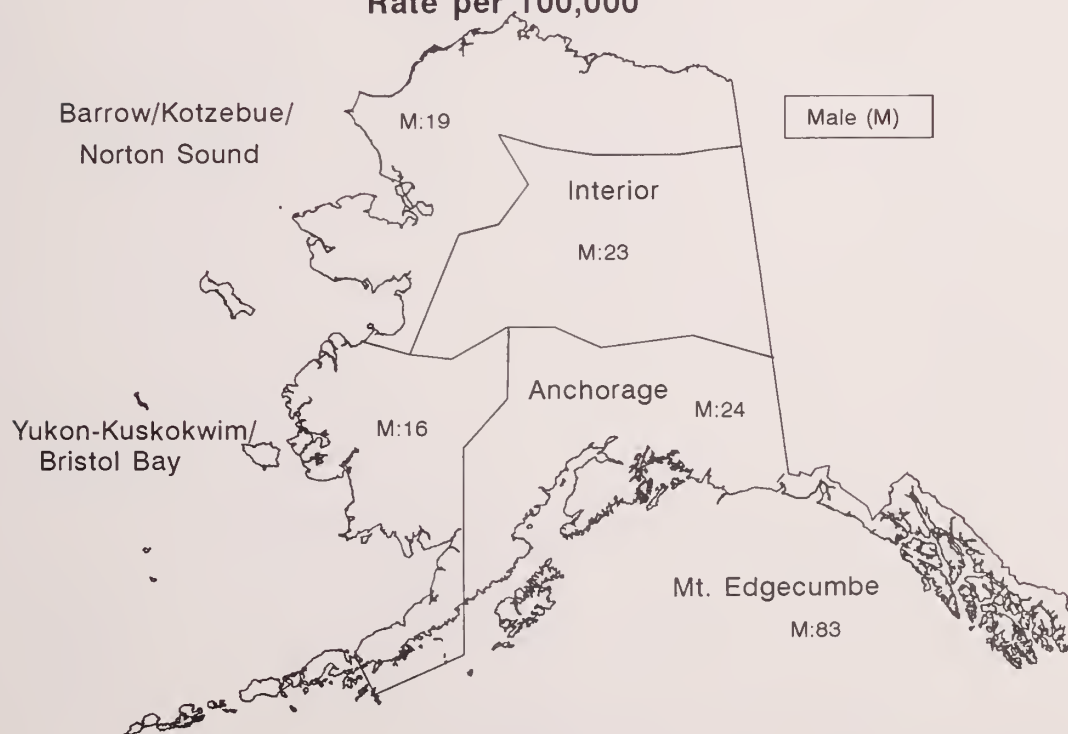
Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

**Prostate**



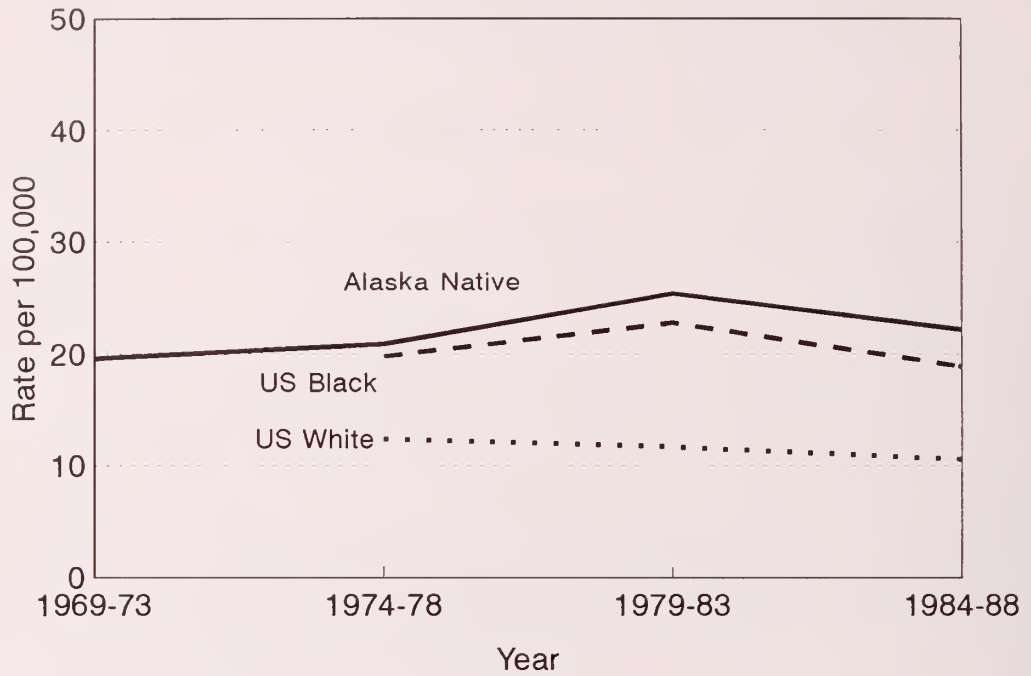
Average Annual Age-Adjusted Cancer Incidence 1969-1988

**Prostate**  
Rate per 100,000

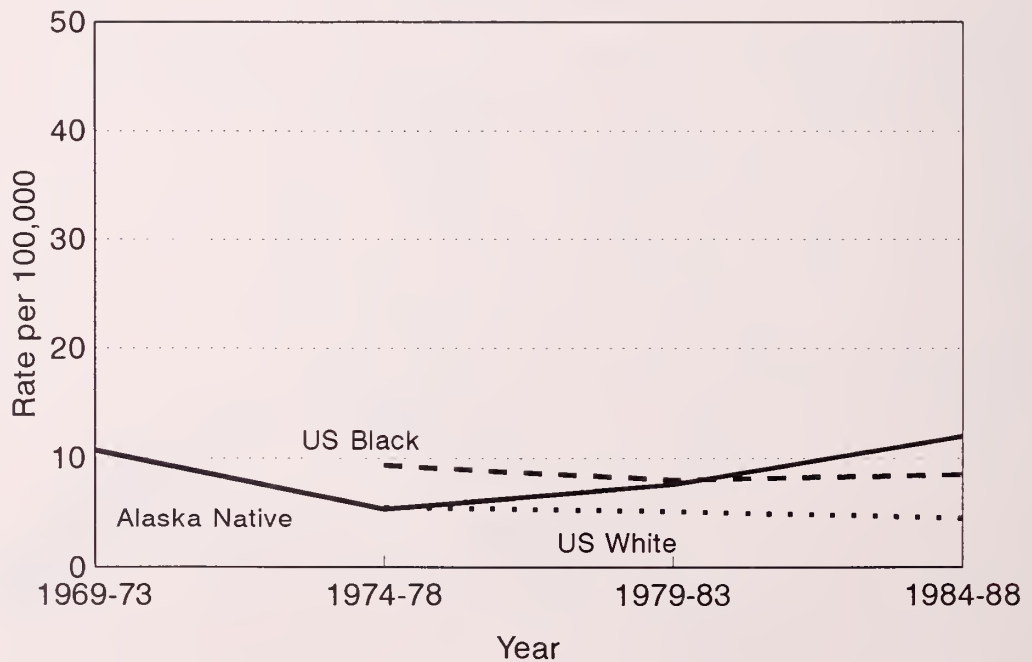


Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US 1974-1988

***Stomach: Male***



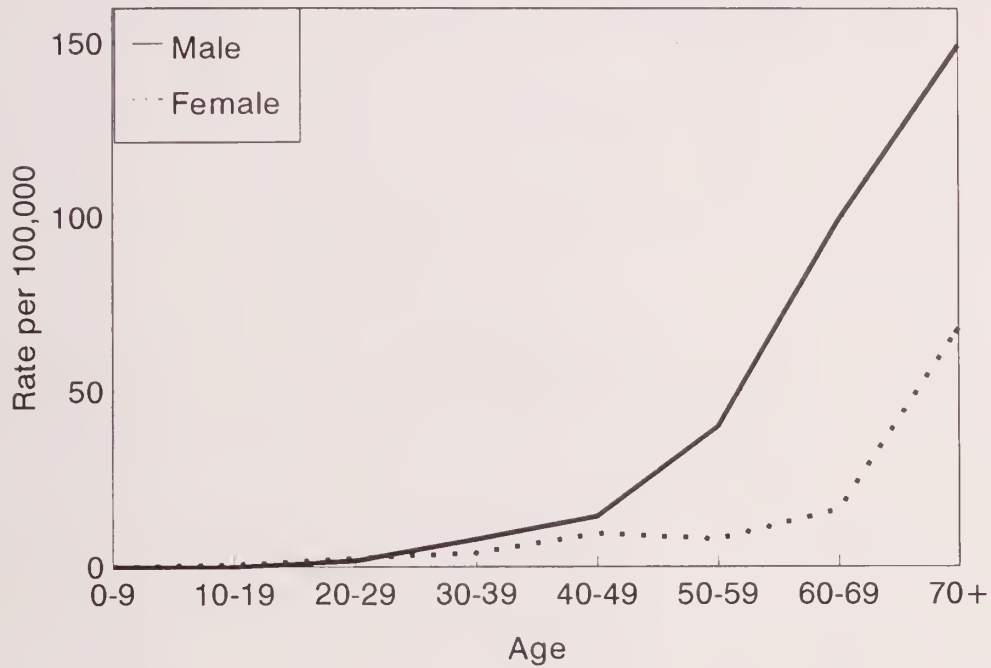
***Stomach: Female***



<sup>1</sup>US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.

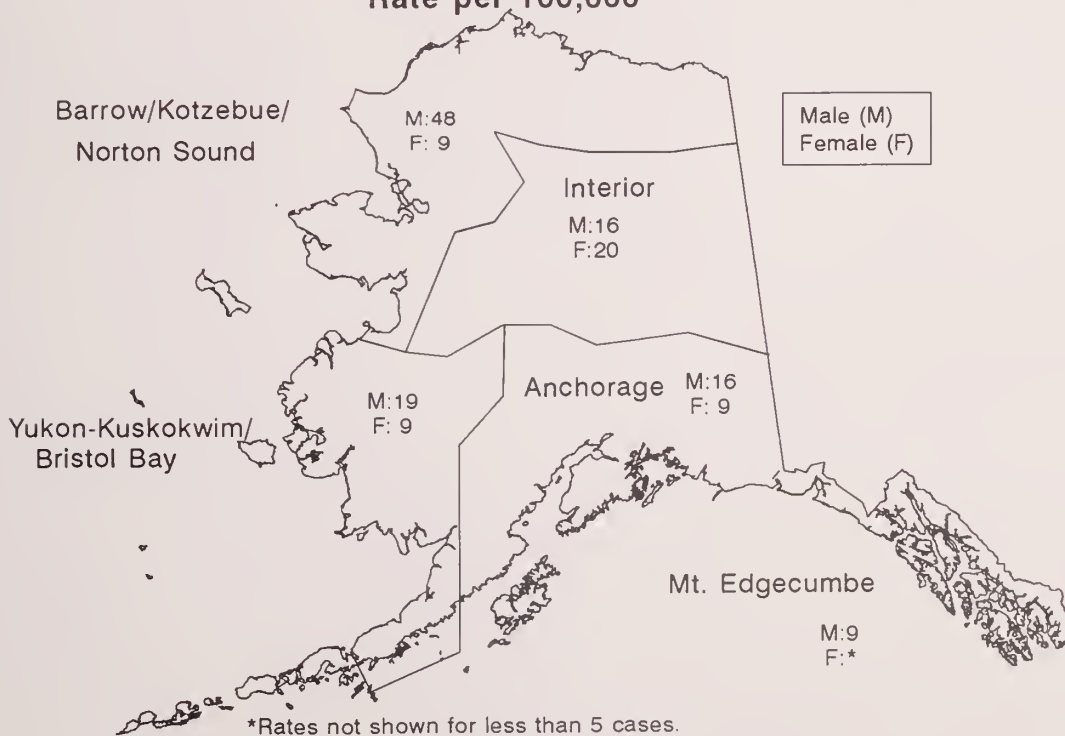
Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

***Stomach***



Average Annual Age-Adjusted Cancer Incidence 1969-1988

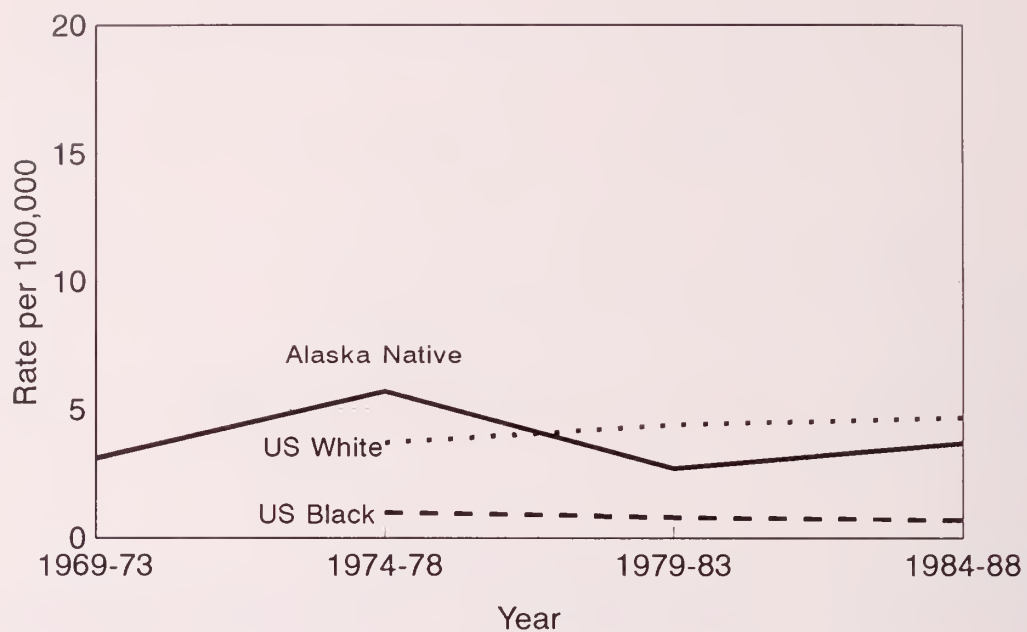
***Stomach***  
**Rate per 100,000**





Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US 1974-1988

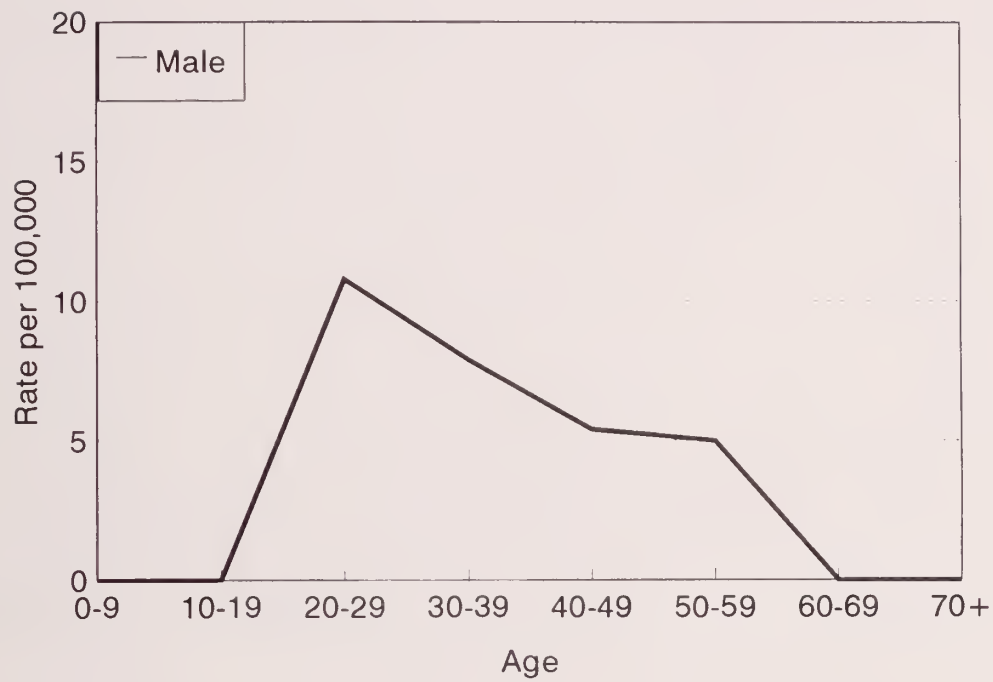
**Testis**



US rates from SEER Program, National Cancer Institute, 1973-1988.  
ALL rates are age-adjusted to 1970 US standard population.

Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

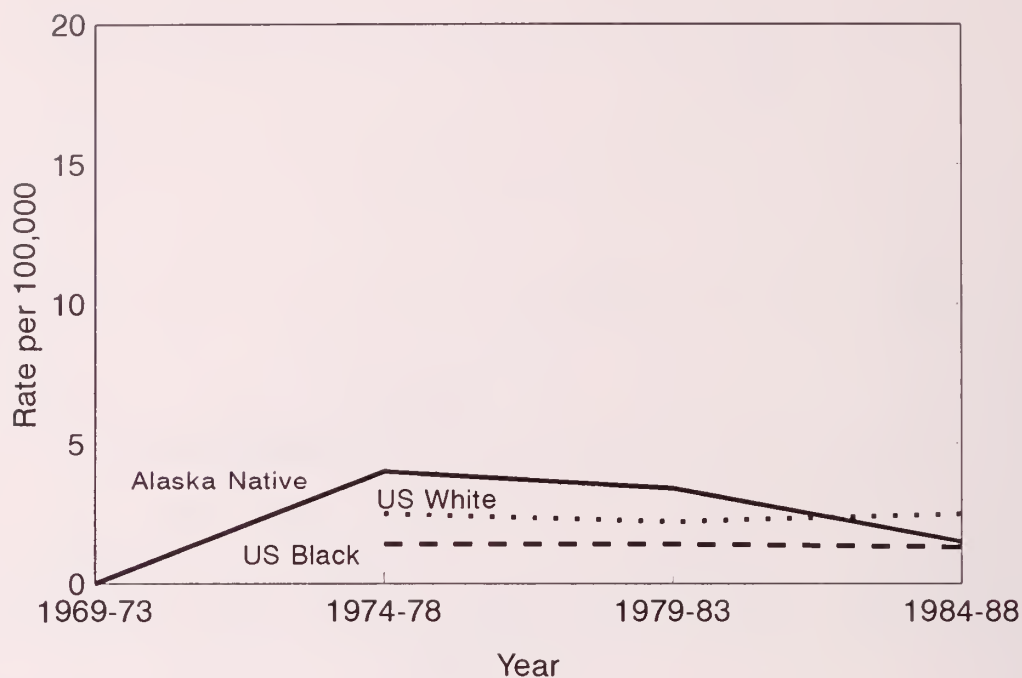
**Testis**



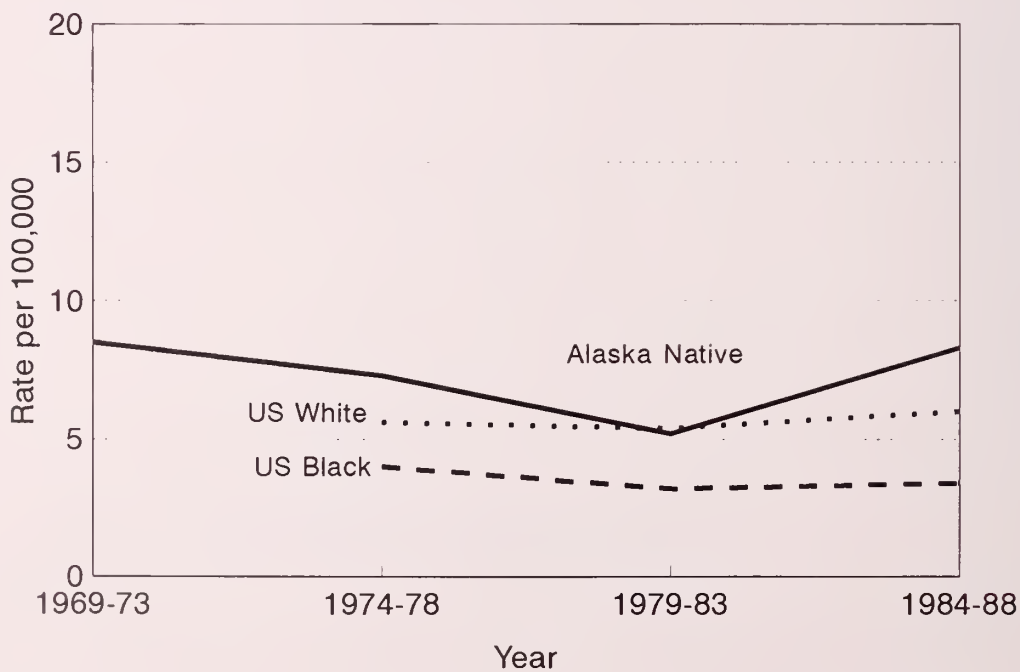
Too few cases to show rates by Service Unit.

Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US 1974-1988

***Thyroid: Male***



***Thyroid: Female***

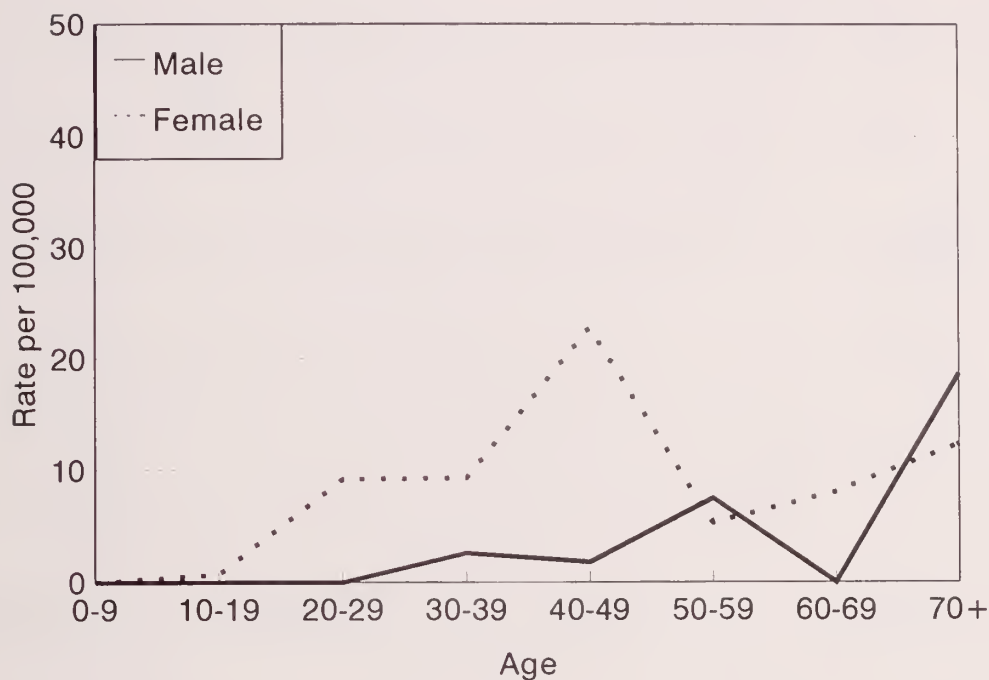


US<sup>1</sup> rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.



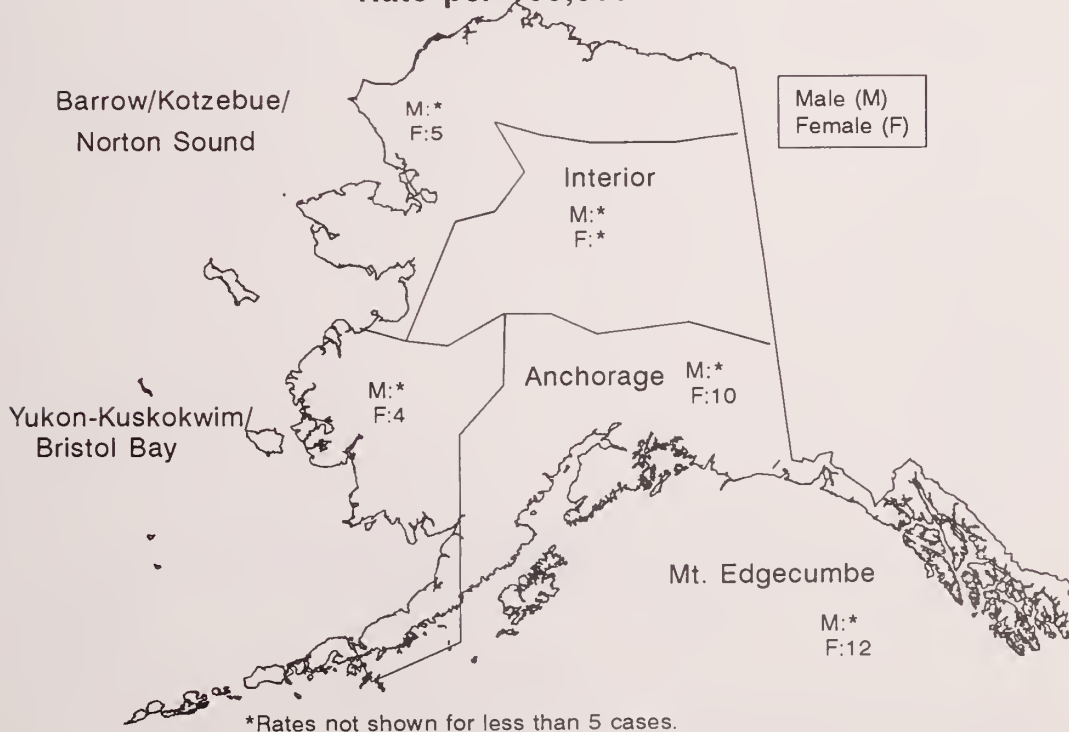
Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

**Thyroid**



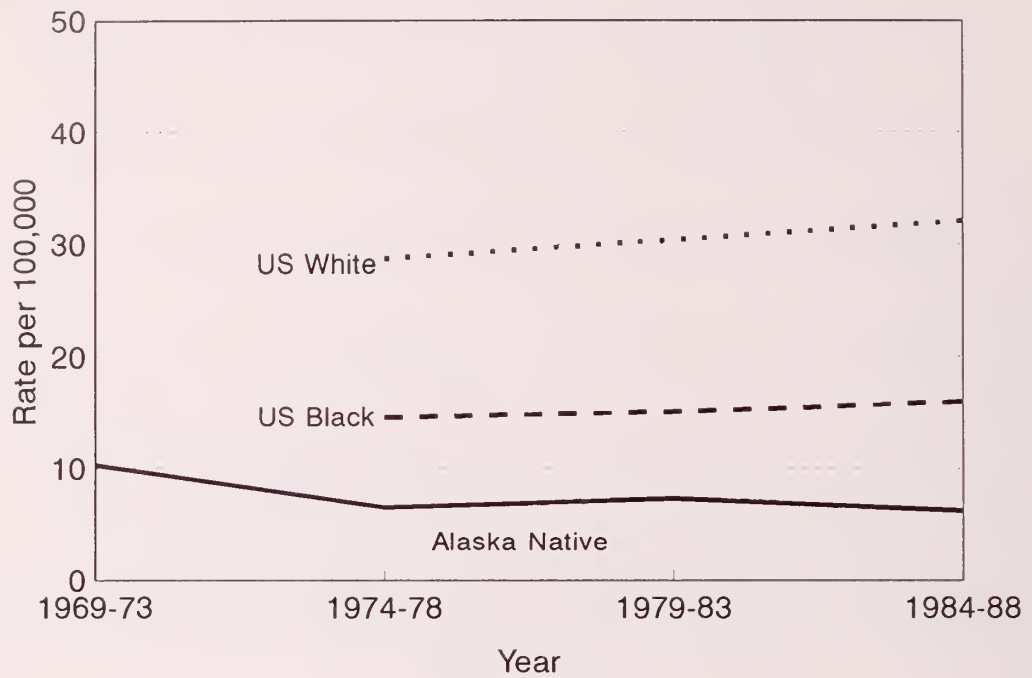
Average Annual Age-Adjusted Cancer Incidence 1969-1988

**Thyroid**  
**Rate per 100,000**

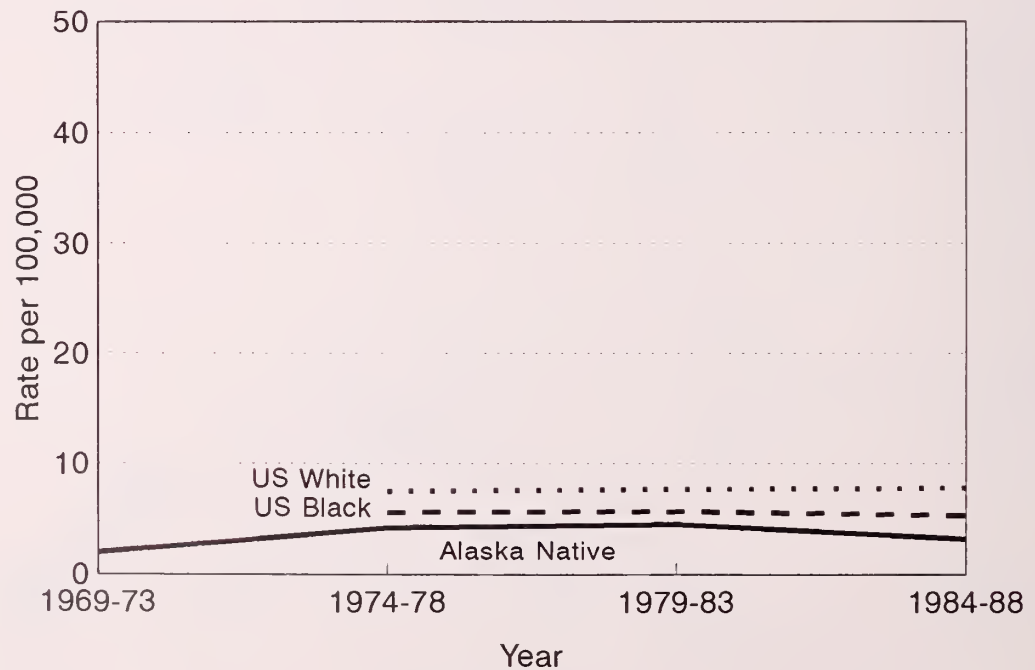


Average Annual Age-Adjusted Cancer Incidence Rates,  
Alaska Natives 1969-1988 and US 1974-1988

**Urinary Bladder: Male**



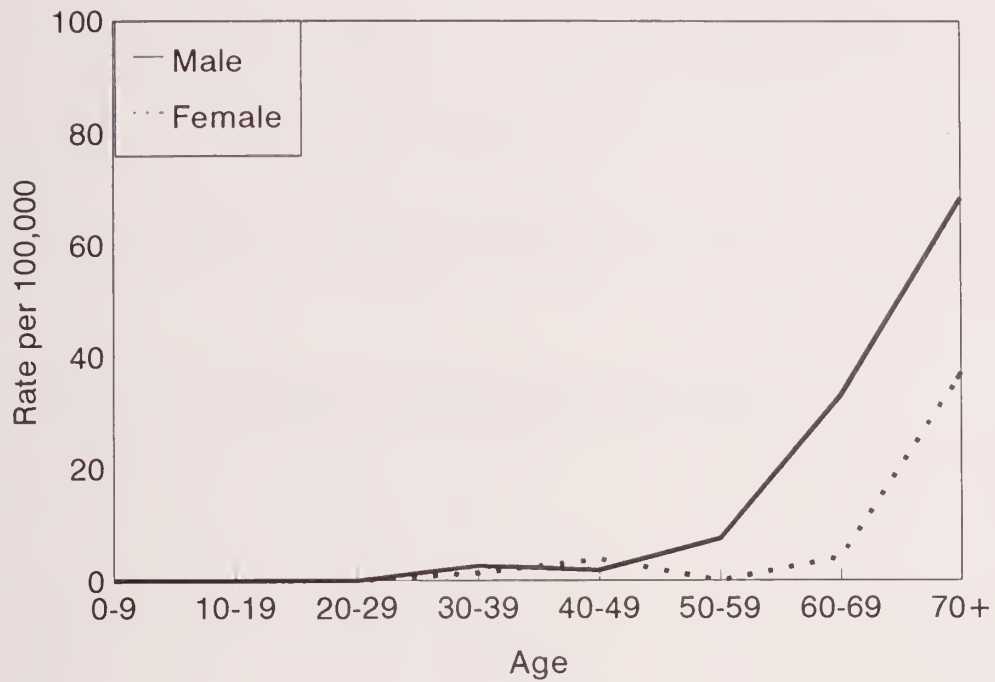
**Urinary Bladder: Female**



<sup>1</sup>US rates from SEER Program, National Cancer Institute, 1973-1988.  
All rates are age-adjusted to 1970 US standard population.

Average Annual Age-Specific Cancer Incidence Rates,  
Alaska Natives 1969-1988

**Urinary Bladder**



Too few cases to show rates by Service Unit.



# DISTRIBUTION OF CANCERS

## 1969-1988

### ALASKA NATIVES

This section presents the twenty year cancer data as percents of total and rank order. The first table shows the proportion of cancers diagnosed at each decade of age as a percent of the total cases identified over the twenty year period.

The other tables show cancer sites in rank order by sex and ethnicity, and by age. These data are included for completeness. Please note that the rank order given here for the twenty year period differs from that presented in the Project Summary. The Project Summary discussed

rank order for the years 1984-88, which best represents the *current* situation. Breast cancer is now the most frequently diagnosed invasive cancer in women, and lung cancer is the most frequently occurring cancer in both sexes combined.

Note the numbers and types of cancers with which tobacco use is associated. These are PREVENTABLE. However, these cancers will continue to occur at the same or higher rates unless tobacco use is markedly reduced.

#### Invasive Cancers in Alaska Natives 1969-1988 Percentage of Total Cases for All Sites, by Age, Both Sexes Combined

Age	# Cases	% of Total Cases
0-9	38	1.6
10-19	41	1.8
20-29	90	3.9
30-39	131	5.7
40-49	279	12.0
50-59	465	20.1
60-69	575	24.8
70+	699	30.2

#### Percentage of Total Cases for All Sites, by Age, Males

Age	# Male Cases	% of Male Cases	% of Total Cases
0-9	23	2.0	1.0
10-19	22	1.9	1.0
20-29	31	2.7	1.3
30-39	44	3.9	1.9
40-49	103	9.0	4.4
50-59	222	19.5	9.6
60-69	324	28.4	14.0
70+	372	32.6	16.1
Total	1141	100.0	49.2

#### Percentage of Total Cases for All Sites, by Age, Females

Age	# Female Cases	% of Female Cases	% of Total Cases
0-9	15	1.3	0.7
10-19	19	1.6	0.8
20-29	59	5.0	2.6
30-39	87	7.4	3.8
40-49	176	15.0	7.6
50-59	243	20.7	10.5
60-69	251	21.3	10.8
70+	327	27.8	14.1
Total	1177	100.0	50.8

## Invasive Cancers in Alaska Natives 1969-1988 Rank Order by Sex

### Male & Female

No. of Cases	Rank
470	Colon/Rectum
402	<b>Lung/Bronchus*</b>
210	Breast
122	<b>Oral/Pharynx*</b>
110	Stomach
109	<b>Cervix*</b>
96	Prostate
96	<b>Kidney/Renal Pelvis*</b>

### Male

No. of Cases	Rank
252	<b>Lung/Bronchus*</b>
193	Colon/Rectum
96	Prostate
80	Stomach
68	<b>Oral/Pharynx*</b>
46	<b>Kidney/Renal Pelvis*</b>
43	Nasopharynx <sup>+</sup>
43	Liver

### Female

No. of Cases	Rank
218	Colon/Rectum
209	Breast
127	<b>Lung/Bronchus*</b>
109	<b>Cervix*</b>
54	<b>Oral/Pharynx*</b>
50	<b>Kidney/Renal Pelvis*</b>
40	Ovary

## Invasive Cancers in Alaska Natives 1969-1988 Rank Order by Ethnicity and Sex: Males

### Aleut

No. of Cases	Rank
32	<b>Lung/Bronchus*</b>
28	Colon/Rectum
14	<b>Oral/Pharynx*</b>
13	Prostate
11	Stomach
10	Nasopharynx <sup>+</sup>
8	Lymphoma

### Indian

No. of Cases	Rank
76	<b>Lung/Bronchus*</b>
67	Colon/Rectum
57	Prostate
20	<b>Kidney/Renal Pelvis*</b>
18	<b>Oral/Pharynx*</b>
11	Leukemia
11	Lymphoma
11	Stomach

### Eskimo

No. of Cases	Rank
144	<b>Lung/Bronchus*</b>
98	Colon/Rectum
58	Stomach
36	<b>Oral/Pharynx*</b>
36	Liver
27	Nasopharynx <sup>+</sup>
26	Prostate
21	<b>Pancreas*</b>
21	<b>Kidney/Renal Pelvis*</b>

## Rank Order by Ethnicity and Sex: Females

### Aleut

No. of Cases	Rank
42	Colon/Rectum
26	Breast
26	<b>Lung/Bronchus*</b>
18	<b>Cervix*</b>
12	<b>Pancreas*</b>
7	<b>Oral/Pharynx*</b>
7	Ovary
6	Thyroid

### Indian

No. of Cases	Rank
105	Breast
58	Colon/Rectum
45	<b>Lung/Bronchus*</b>
39	<b>Cervix*</b>
20	<b>Kidney/Renal Pelvis*</b>
18	<b>Oral/Pharynx*</b>
18	Ovary
17	Thyroid

### Eskimo

No. of Cases	Rank
118	Colon/Rectum
78	Breast
56	<b>Lung/Bronchus*</b>
52	<b>Cervix*</b>
29	<b>Oral/Pharynx*</b>
25	<b>Kidney/Renal Pelvis*</b>
24	Gallbladder
15	Ovary
15	Nasopharynx <sup>+</sup>
15	Stomach
15	<b>Pancreas*</b>

**\* TOBACCO USE IS A RISK FACTOR FOR THESE CANCERS.**

+ Cancers of the Nasopharynx were also included in the Oral/Pharynx category.

# Invasive Cancers in Alaska Natives 1969-1988

## Rank Order by Age and Sex: Males

### Ages 0-9

No. of Cases	Rank
5	Brain/Nervous Sys.
4	Leukemia
3	Lymphoma
2	Liver
2	Eye/Orbit

### Ages 10-19

No. of Cases	Rank
6	Liver
5	Leukemia
2	Lymphoma
2	Brain/Nervous Sys.
2	Soft Tissue

### Ages 20-29

No. of Cases	Rank
13	Testis
5	Liver
2	Leukemia
2	Lymphoma
2	Brain/Nervous Sys.
2	Stomach
2	Colon/Rectum

### Ages 30-39

No. of Cases	Rank
6	Testis
6	Stomach
4	<b>Oral/Pharynx*</b>
3	Nasopharynx <sup>+</sup>
3	Leukemia
3	Colon/Rectum

### Ages 40-49

No. of Cases	Rank
19	<b>Oral/Pharynx*</b>
14	Colon/Rectum
12	Nasopharynx <sup>+</sup>
11	<b>Lung/Bronchus*</b>
8	Stomach
7	<b>Pancreas*</b>
6	Liver
5	Lymphoma

### Ages 50-59

No. of Cases	Rank
66	<b>Lung/Bronchus*</b>
36	Colon/Rectum
22	<b>Oral/Pharynx*</b>
16	Stomach
13	Nasopharynx <sup>+</sup>
10	<b>Kidney/Renal Pelvis*</b>
8	<b>Pancreas*</b>
8	Liver
8	Prostate

### Ages 60-69

No. of Cases	Rank
100	<b>Lung/Bronchus*</b>
54	Colon/Rectum
31	Prostate
24	Stomach
16	<b>Kidney/Renal Pelvis*</b>
15	<b>Oral/Pharynx*</b>
12	Nasopharynx <sup>+</sup>
12	<b>Pancreas*</b>
9	Liver
8	<b>Urinary Bladder*</b>

### Ages 70+

No. of Cases	Rank
83	Colon/Rectum
73	<b>Lung/Bronchus*</b>
56	Prostate
24	Stomach
15	<b>Esophagus*</b>
15	<b>Kidney/Renal Pelvis*</b>
13	Lymphoma
11	<b>Urinary Bladder*</b>

**\* TOBACCO USE IS A RISK FACTOR ASSOCIATED WITH THESE CANCERS.**

+ Cancers of the Nasopharynx were also included in the Oral/Pharynx category.



# Invasive Cancers in Alaska Natives 1969-1988

## Rank Order by Age and Sex: Females

### Ages 0-9

No. of Cases	Rank
5	Brain/Nervous Sys.
3	Eye/Orbit
2	Leukemia
2	<b>Kidney/Renal Pelvis*</b>

### Ages 10-19

No. of Cases	Rank
3	Brain/Nervous Sys.
3	Liver
2	Leukemia
2	<b>Cervix*</b>
2	Ovary
2	Bones/Joints

### Ages 20-29

No. of Cases	Rank
16	<b>Cervix*</b>
11	Thyroid
6	Breast
6	Ovary
3	Lymphoma
3	Stomach
2	Leukemia

### Ages 30-39

No. of Cases	Rank
28	<b>Cervix*</b>
23	Breast
7	Thyroid
4	<b>Lung/Bronchus*</b>
4	<b>Oral/Pharynx*</b>
3	Stomach
3	Colon/Rectum

### Ages 40-49

No. of Cases	Rank
60	Breast
23	<b>Cervix*</b>
19	<b>Lung/Bronchus*</b>
15	Colon/Rectum
14	<b>Oral/Pharynx*</b>
12	Thyroid
7	Nasopharynx <sup>+</sup>
7	<b>Kidney/Renal Pelvis*</b>
5	Ovary
5	Stomach

### Ages 50-59

No. of Cases	Rank
62	Breast
37	Colon/Rectum
28	<b>Lung/Bronchus*</b>
19	<b>Cervix*</b>
14	Ovary
12	<b>Kidney/Renal Pelvis*</b>
9	<b>Pancreas*</b>
7	Corpus uteri/NOS
7	<b>Oral/Pharynx*</b>

### Ages 60-69

No. of Cases	Rank
64	Colon/Rectum
38	<b>Lung/Bronchus*</b>
29	Breast
20	<b>Oral/Pharynx*</b>
15	<b>Pancreas*</b>
13	<b>Cervix*</b>
12	Nasopharynx <sup>+</sup>
10	Gallbladder
9	<b>Kidney/Renal Pelvis*</b>

### Ages 70+

No. of Cases	Rank
97	Colon/Rectum
37	<b>Lung/Bronchus*</b>
29	Breast
16	<b>Kidney/Renal Pelvis*</b>
11	Stomach
8	<b>Cervix*</b>
8	<b>Oral/Pharynx*</b>
7	<b>Pancreas*</b>

**\* TOBACCO USE IS A RISK FACTOR FOR THESE CANCERS.**

+Cancers of the Nasopharynx were also included in the Oral/Pharynx category.

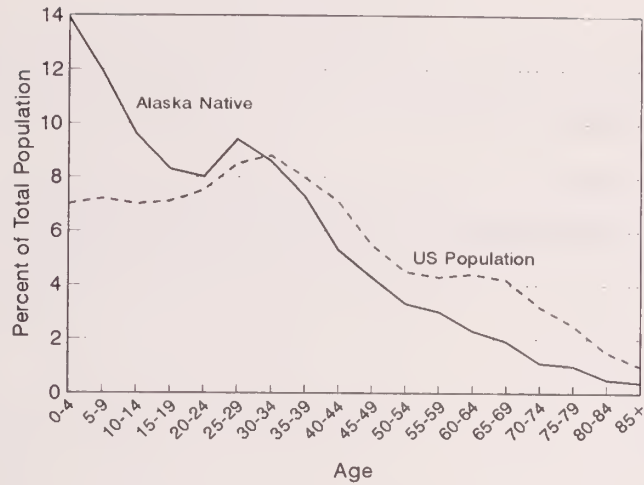
## ALASKA NATIVE POPULATION

This section presents some facts about the Alaska Native population in graphic format. The map below shows the areas of the state originally inhabited by various Alaska Native groups. The next page shows: the age distribution of the Alaska Native population compared with the U.S. in 1990; the Alaska Native population for 1980, 1990, and 2000 (projected); and the percent of the total Native population represented by each ethnic group. The U.S. population as a whole has a much larger proportion of people in the over 35 age

group, i.e. in those at greatest risk for developing cancer. Note the progressive increase in the Native population in the older age groups over time. For a disease such as cancer, this can only mean an increase in the numbers of patients diagnosed with cancer each year. Numbers of cases would increase just from the effect of the shift in the age distribution of the population. However, if the actual rates of cancer are also increasing, as they are dramatically in the Alaska Native population, the yearly increase in numbers of patients with cancer will be magnified.

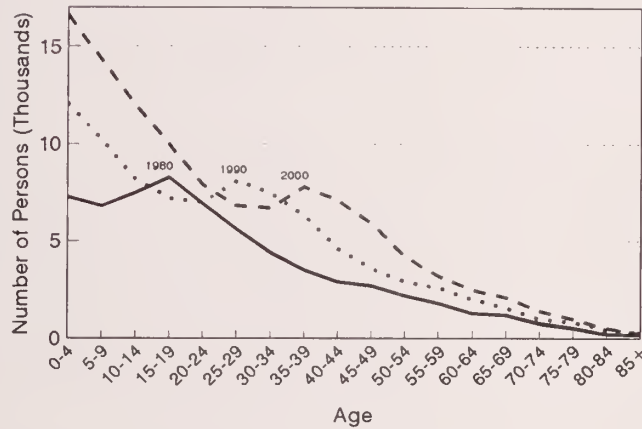


# Alaska Native and US Populations 1990



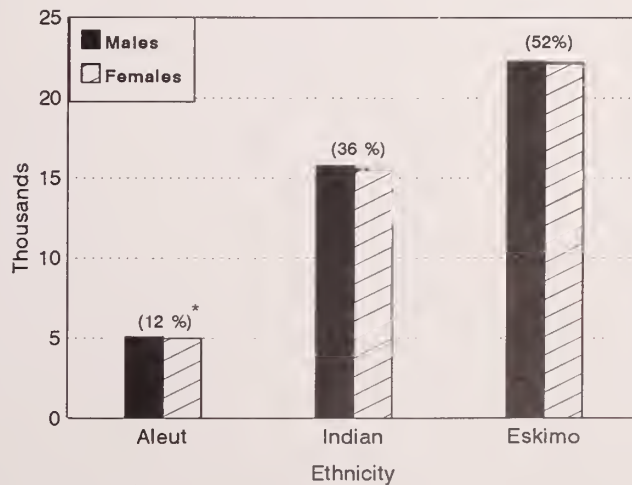
Source: Census 1990

## Alaska Native Population 1980, 1990, and Population Projection Year 2000



Source: Boedeker B. and Butler S. Trends in Native Death Rates. Division of Planning, Evaluation & Health Statistics, Office of Patient Care Standards, Alaska Area Native Health Service, Anchorage, Alaska, August 28, 1993

## Native Population by Ethnicity 1990



\* Percent of Alaska Native Population.

Source: 1990 Census



## APPENDIX

The appendix includes detailed cancer case counts and populations used to develop the tables, graphs and maps presented in this report. International Classification of Disease for Oncology (ICD-O-2) codes for cancer sites and site groups have also been included.

### Invasive Cancers in Alaska Natives 1969-1988 Average Annual Incidence Rates per 100,000, Males

Site	Crude Rate	Age-Adjusted* Rate
All Sites	178.9	315.2
Oral Cavity and Pharynx	10.7	17.0
Salivary Gland	0.8	1.1
Nasopharynx	6.7	10.9
Gum & Other Oral Cavity	1.1	1.5
Digestive	64.0	115.4
Esophagus	3.9	7.7
Stomach	12.5	22.1
Colon & Rectum	30.3	56.7
Colon	21.0	39.6
Rectosigmoid Junction	2.7	4.6
Rectum	6.6	12.6
Liver	6.7	9.6
Gallbladder	1.6	3.2
Other Biliary	1.6	3.0
Pancreas	5.7	9.9
Respiratory	42.0	77.5
Lung & Bronchus	39.5	73.6
Larynx	1.1	1.7
Bones & Joints	1.6	2.2
Soft Tissue	1.6	2.3
Melanoma of Skin	0.3	0.4
Breast	0.2	0.3
Female Genital	-	-
Cervix uteri	-	-
Corpus uteri/Uterus, NOS	-	-
Ovary	-	-
Male Genital	20.1	35.5
Prostate	15.1	30.2
Testis	3.9	3.7
Urinary	11.3	21.0
Bladder	3.9	7.3
Kidney & Renal Pelvis	7.2	13.3
Eye & Orbit	0.5	0.5
Brain & Nervous System	2.4	2.6
Brain	2.4	2.6
Endocrine	2.2	3.4
Thyroid	1.4	2.3
Lymphomas	5.7	9.1
Hodgkin's	0.6	0.6
Non-Hodgkin's	5.0	8.5
Multiple Myeloma	2.0	3.9
Leukemia	3.9	4.7
Ill Defined & Unspecified	10.2	18.5

\*Rates are age-adjusted to 1970 US standard population.

**Invasive Cancers in Alaska Natives 1969-1988**  
**Average Annual Incidence Rates per 100,000, Females**

Site	Crude Rate	Age-Adjusted* Rate
All Sites	190.5	365.3
Oral Cavity and Pharynx	8.7	16.1
Salivary Gland	1.9	2.7
Nasopharynx	3.6	6.4
Gum & Other Oral Cavity	1.1	2.7
Digestive	58.3	129.4
Esophagus	1.9	4.4
Stomach	4.9	9.3
Colon & Rectum	35.3	80.3
Colon	27.5	62.2
Rectosigmoid Junction	2.6	6.2
Rectum	5.2	11.8
Liver	1.8	3.3
Gallbladder	5.5	13.7
Other Biliary	0.7	1.0
Pancreas	5.5	11.3
Respiratory	21.5	44.1
Lung & Bronchus	20.6	42.4
Larynx	0.7	1.0
Bones & Joints	1.3	2.0
Soft Tissue	0.8	1.9
Melanoma of Skin	0.8	1.6
Breast	33.8	57.9
Female Genital	28.3	42.2
Cervix uteri	17.6	24.8
Corpus uteri/Uterus, NOS	2.9	4.6
Ovary	6.5	10.6
Male Genital	-	-
Prostate	-	-
Testis	-	-
Urinary	9.9	20.0
Bladder	1.6	3.7
Kidney & Renal Pelvis	8.1	16.1
Eye & Orbit	0.5	0.4
Brain & Nervous System	1.9	2.0
Brain	1.8	1.8
Endocrine	6.0	7.4
Thyroid	6.0	7.4
Lymphomas	2.9	5.3
Hodgkin's	0.7	1.0
Non-Hodgkin's	2.3	4.2
Multiple Myeloma	0.8	1.5
Leukemia	2.3	3.5
III Defined & Unspecified	12.5	29.8

\*Rates are age-adjusted to 1970 US standard population.

**Invasive Cancers in  
Alaska Natives 1969-1988  
Number of Cases, Males**

Site	1969-73	1974-78	1979-83	1984-88	Total
All Sites	207	251	308	375	1141
Oral Cavity & Pharynx	14	22	9	23	68
Salivary Gland	3	0	1	1	5
Nasopharynx	9	17	6	11	43
Gum & Other Oral Cavity	0	2	2	3	7
Digestive	77	88	111	132	408
Esophagus	6	5	7	7	25
Stomach	12	17	25	26	80
Colon & Rectum	36	45	46	66	193
Colon	22	29	36	47	134
Rectosigmoid Junction	3	6	3	5	17
Rectum	11	10	7	14	42
Liver	9	10	13	11	43
Gallbladder	2	2	5	1	10
Other Biliary	2	1	0	7	10
Pancreas	8	7	9	12	36
Respiratory	36	57	73	102	268
Lung & Bronchus	34	55	66	97	252
Larynx	0	1	5	1	7
Bones & Joints	4	1	0	5	10
Soft Tissue	4	4	0	2	10
Melanoma of Skin	0	1	1	0	2
Breast	0	0	1	0	1
Female Genital	-	-	-	-	-
Cervix uteri	-	-	-	-	-
Corpus uteri/Uterus, NOS	-	-	-	-	-
Ovary	-	-	-	-	-
Male Genital	23	23	41	41	128
Prostate	16	14	35	31	96
Testis	4	8	5	8	25
Urinary	15	16	14	27	72
Bladder	7	5	6	7	25
Kidney & Renal Pelvis	8	10	8	20	46
Eye & Orbit	0	0	3	0	3
Brain & Nervous System	3	5	3	4	15
Brain	3	5	3	4	15
Endocrine	0	5	5	4	14
Thyroid	0	4	3	2	9
Lymphomas	5	4	18	9	36
Hodgkin's	0	1	2	1	4
Non-Hodgkin's	5	3	16	8	32
Multiple Myeloma	4	3	4	2	13
Leukemia	7	5	5	8	25
Ill Defined & Unspecified	15	17	20	16	68



**Invasive Cancers in  
Alaska Natives 1969-1988  
Number of Cases, Females**

Site	1969-73	1974-78	1979-83	1984-88	Total
All Sites	187	235	301	454	1177
Oral Cavity & Pharynx	13	12	15	14	54
Salivary Gland	5	3	1	3	12
Nasopharynx	3	7	6	6	22
Gum & Other Oral Cavity	2	0	4	1	7
Digestive	67	67	90	136	360
Esophagus	1	3	3	5	12
Stomach	6	4	6	14	30
Colon & Rectum	37	39	56	86	218
Colon	25	32	43	70	170
Rectosigmoid Junction	3	2	6	5	16
Rectum	9	5	7	11	32
Liver	1	3	2	5	11
Gallbladder	9	10	7	8	34
Other Biliary	3	0	0	1	4
Pancreas	5	7	9	13	34
Respiratory	9	15	29	80	133
Lung & Bronchus	9	14	29	75	127
Larynx	0	0	0	4	4
Bones & Joints	0	2	2	4	8
Soft Tissue	0	1	2	2	5
Melanoma of Skin	1	1	1	2	5
Breast	26	42	49	92	209
Female Genital	24	40	58	53	175
Cervix uteri	12	27	42	28	109
Corpus uteri/Uterus, NOS	3	3	5	7	18
Ovary	8	9	8	15	40
Male Genital	-	-	-	-	-
Prostate	-	-	-	-	-
Testis	-	-	-	-	-
Urinary	11	12	12	26	61
Bladder	2	2	3	3	10
Kidney & Renal Pelvis	9	10	8	23	50
Eye & Orbit	1	0	1	1	3
Brain & Nervous System	4	2	2	4	12
Brain	3	2	2	4	11
Endocrine	10	8	8	11	37
Thyroid	10	8	8	11	37
Lymphomas	2	4	5	7	18
Hodgkin's	0	0	2	2	4
Non-Hodgkin's	2	4	3	5	14
Multiple Myeloma	2	0	1	2	5
Leukemia	3	3	3	5	14
Ill Defined & Unspecified	14	26	23	15	78

**Invasive Cancers in  
Alaska Natives 1969-1988  
Number of Cases by Age, Males**

Site	0-9	10-19	20-29	Age 30-39	40-49	50-59	60-69	70+
All Sites	23	22	31	44	103	222	324	372
Oral Cavity & Pharynx	0	0	2	4	19	22	15	6
Salivary Gland	0	0	1	0	1	2	1	0
Nasopharynx	0	0	0	3	12	13	12	3
Gum & Other Oral Cavity	0	0	1	1	2	3	0	0
Digestive System	2	7	9	12	39	74	116	149
Esophagus	0	0	0	0	2	2	6	15
Stomach	0	0	2	6	8	16	24	24
Colon & Rectum	0	1	2	3	14	36	54	83
Colon	0	0	2	2	8	25	38	59
Rectosigmoid Junction	0	0	0	1	4	4	3	5
Rectum	0	1	0	0	2	7	13	19
Liver	2	6	5	2	6	8	9	5
Gallbladder	0	0	0	0	0	1	2	7
Other Biliary	0	0	0	0	0	2	4	4
Pancreas	0	0	0	1	7	8	12	8
Respiratory System	0	0	0	4	16	71	102	75
Lung & Bronchus	0	0	0	2	11	66	100	73
Larynx	0	0	0	0	3	3	1	0
Bones & Joints	1	0	1	2	1	1	3	1
Soft Tissue	0	2	0	2	0	3	0	3
Melanoma of Skin	0	0	0	1	1	0	0	0
Breast	0	0	0	0	0	0	1	0
Male Genital	3	0	13	6	5	12	32	57
Prostate	0	0	0	0	1	8	31	56
Testis	1	0	13	6	3	2	0	0
Urinary	1	0	0	2	5	13	24	27
Bladder	0	0	0	2	1	3	8	11
Kidney & Renal Pelvis	1	0	0	0	4	10	16	15
Eye & Orbit	2	0	0	0	0	1	0	0
Brain & Nervous System	5	2	2	1	3	0	1	1
Brain	5	2	2	1	3	0	1	1
Endocrine	0	1	0	2	2	5	1	3
Thyroid	0	0	0	2	1	3	0	3
Lymphomas	3	2	2	2	5	3	6	13
Hodgkin's	1	0	1	1	1	0	0	0
Non-Hodgkin's	2	2	1	1	4	3	6	13
Multiple Myeloma	1	2	0	0	1	3	3	7
Leukemia	4	5	2	3	1	1	1	4
Ill Defined & Unspecified	1	1	0	3	5	13	19	26

**Invasive Cancers in  
Alaska Natives 1969-1988  
Number of Cases by Age, Females**

Site	0-9	10-19	20-29	Age 30-39	40-49	50-59	60-69
All Sites	15	19	59	87	176	243	251
Oral Cavity & Pharynx	0	0	1	4	14	7	20
Salivary Gland	0	0	1	4	3	2	1
Nasopharynx	0	0	0	0	7	3	12
Gum & Other Oral Cavity	0	0	0	0	1	0	2
Digestive System	0	4	6	9	24	62	103
Esophagus	0	0	0	0	0	4	3
Stomach	0	1	3	3	5	3	4
Colon & Rectum	0	0	2	3	15	37	64
Colon	0	0	2	2	12	28	53
Rectosigmoid Junction	0	0	0	0	2	1	4
Rectum	0	0	0	1	1	8	7
Liver	0	3	1	0	0	1	2
Gallbladder	0	0	0	0	0	4	10
Other Biliary	0	0	0	1	1	1	1
Pancreas	0	0	0	1	2	9	15
Respiratory System	0	0	1	5	20	29	41
Lung & Bronchus	0	0	1	4	19	28	38
Larynx	0	0	0	1	1	1	1
Bones & Joints	1	2	1	0	0	2	0
Soft Tissue	0	0	0	0	1	1	0
Melanoma of Skin	0	0	0	0	1	1	2
Breast	0	0	6	23	60	62	29
Female Genital System	1	4	26	33	31	41	22
Cervix uteri	0	2	16	28	23	19	13
Corpus uteri/Uterus, NOS	0	0	2	2	3	7	3
Ovary	1	2	6	1	5	14	6
Urinary	2	1	1	3	9	13	10
Bladder	0	0	0	1	2	0	1
Kidney & Renal Pelvis	2	1	1	2	7	12	9
Eye & Orbit	3	0	0	0	0	0	0
Brain & Nervous System	5	3	0	1	0	2	1
Brain	5	3	0	1	0	1	1
Endocrine	0	1	11	7	12	2	2
Thyroid	0	1	11	7	12	2	2
Lymphomas	0	2	3	0	4	2	3
Hodgkin's	0	0	2	0	0	0	1
Non-Hodgkin's	0	1	1	0	4	2	2
Multiple Myeloma	0	0	0	1	0	4	2
Leukemia	2	2	2	0	0	1	2
Ill Defined & Unspecified	1	1	1	1	0	15	14



**Invasive Cancers in  
Alaska Natives 1969-1988  
Number of Cases by IHS Service Units\*, Males**

Site	1	2	3	4	5
All Sites	273	141	181	292	254
Oral Cavity & Pharynx	23	6	5	17	17
Salivary Gland	1	1	0	2	1
Nasopharynx	15	2	2	11	13
Gum & Other Oral Cavity	2	1	1	2	1
Digestive System	91	45	53	115	104
Esophagus	8	2	5	9	1
Stomach	16	6	5	19	34
Colon & Rectum	50	28	29	48	38
Colon	31	18	22	35	28
Rectosigmoid Junction	5	3	3	3	3
Rectum	14	7	4	10	7
Liver	8	2	3	18	12
Gallbladder	0	0	3	3	4
Other Biliary	1	1	3	2	3
Pancreas	7	6	4	11	8
Respiratory System	60	40	24	83	61
Lung & Bronchus	56	38	20	80	58
Larynx	2	1	3	0	1
Bones & Joints	3	3	0	2	2
Soft Tissue	2	2	3	2	1
Melanoma of Skin	0	0	0	1	1
Breast	0	0	1	0	0
Male Genital	34	12	46	18	18
Prostate	21	8	41	14	12
Testis	10	3	4	4	4
Urinary	13	14	12	17	16
Bladder	5	3	6	4	7
Kidney & Renal Pelvis	7	11	6	13	9
Eye & Orbit	1	0	0	0	2
Brain & Nervous System	6	2	2	2	3
Brain	6	2	2	2	3
Endocrine	5	3	3	1	2
Thyroid	2	3	2	1	1
Lymphomas	9	3	8	10	6
Hodgkin's	2	0	0	1	1
Non-Hodgkin's	7	3	8	9	5
Multiple Myeloma	4	0	7	1	1
Leukemia	6	2	8	4	5
Ill Defined & Unspecified	16	9	9	19	15

\* Service Units: 1 = Anchorage, 2 = Interior Alaska, 3 = Mt. Edgecumbe, 4 = Yukon-Kuskowim and Bristol Bay Area,  
5 = Barrow, Kotzebue and Norton Sound

**Invasive Cancers in  
Alaska Natives 1969-1988  
Number of Cases by IHS Service Units\*, Females**

Site	1	2	3	4	5
All Sites	374	149	180	266	207
Oral Cavity & Pharynx	16	6	6	13	13
Salivary Gland	3	4	0	2	3
Nasopharynx	5	0	3	7	7
Gum & Other Oral Cavity	3	0	1	2	1
Digestive System	111	34	40	104	71
Esophagus	2	1	1	6	2
Stomach	8	7	2	7	6
Colon & Rectum	79	19	19	64	37
Colon	59	14	14	53	30
Rectosigmoid Junction	5	2	1	3	5
Rectum	15	3	4	8	2
Liver	2	2	2	1	4
Gallbladder	1	3	5	12	13
Other Biliary	1	0	1	0	2
Pancreas	15	2	4	8	5
Respiratory System	60	14	15	25	18
Lung & Bronchus	55	14	15	24	18
Larynx	4	0	0	0	0
Bones & Joints	2	1	1	3	1
Soft Tissue	2	1	0	0	2
Melanoma of Skin	2	0	2	1	0
Breast	58	32	52	36	31
Female Genital System	62	29	26	25	33
Cervix uteri	32	18	19	15	25
Corpus uteri/Uterus, NOS	3	6	4	2	3
Ovary	23	5	2	6	4
Urinary	16	11	7	21	6
Bladder	8	0	1	1	0
Kidney & Renal Pelvis	8	11	5	20	6
Eye & Orbit	0	1	1	1	0
Brain & Nervous System	2	0	2	4	4
Brain	2	0	1	4	4
Endocrine	14	4	9	5	5
Thyroid	14	4	9	5	5
Lymphomas	6	3	2	4	3
Hodgkin's	2	0	1	1	0
Non-Hodgkin's	4	3	1	3	3
Multiple Myeloma	1	1	2	0	1
Leukemia	2	3	3	6	0
Ill Defined & Unspecified	20	9	12	18	19

\*Service Units: 1 = Anchorage, 2 = Interior Alaska, 3 = Mt. Edgecumbe, 4 = Yukon-Kuskowim and Bristol Bay Area,  
5 = Barrow, Kotzebue and Norton Sound

### 1970 ALASKA NATIVE POPULATION BY ETHNICITY

AGE	<u>ALEUT</u>			<u>INDIAN</u>			<u>ESKIMO</u>			<u>TOTAL</u>		
	MALES	FEMALES	TOTAL	MALES	FEMALES	TOTAL	MALES	FEMALES	TOTAL	MALES	FEMALES	TOTAL
0-4	350	382	732	1115	882	1997	2112	1933	4045	3577	3197	6774
5-9	456	453	909	1317	1149	2466	2469	2289	4758	4242	3891	8133
10-14	522	424	946	1145	1192	2337	2174	2117	4291	3841	3733	7574
15-19	406	297	703	793	866	1659	1504	1757	3261	2703	2920	5623
20-24	279	273	552	504	612	1116	935	1054	1989	1718	1939	3657
25-29	220	157	377	703	618	1321	874	894	1768	1797	1669	3466
30-34	241	251	492	551	394	945	766	815	1581	1558	1460	3018
35-39	167	143	310	394	447	841	610	640	1250	1171	1230	2401
40-44	183	145	328	463	345	808	595	619	1214	1241	1109	2350
45-49	155	131	286	296	299	595	588	434	1022	1039	864	1903
50-54	146	104	250	255	274	529	399	444	843	800	822	1622
55-59	53	83	136	267	249	516	451	373	824	771	705	1476
60-64	91	77	168	211	173	384	219	199	418	521	449	970
65-69	53	54	107	193	143	336	304	139	443	550	336	886
70-74	5	16	21	93	83	176	83	65	148	181	164	345
75+	32	3	35	122	133	255	122	209	331	276	345	621
Total	3359	2993	6352	8422	7859	16281	14205	13981	28186	25986	24833	50819

### 1980 ALASKA NATIVE POPULATION BY ETHNICITY

AGE	<u>ALEUT</u>			<u>INDIAN</u>			<u>ESKIMO</u>			<u>TOTAL</u>		
	MALES	FEMALES	TOTAL	MALES	FEMALES	TOTAL	MALES	FEMALES	TOTAL	MALES	FEMALES	TOTAL
0-4	413	402	815	1301	1172	2473	2066	1966	4032	3780	3540	7320
5-9	397	382	779	1088	1138	2226	1992	1870	3862	3477	3390	6867
10-14	460	454	914	1277	1174	2451	2120	2010	4130	3857	3638	7495
15-19	533	517	1050	1390	1358	2748	2332	2216	4548	4255	4091	8346
20-24	460	455	915	1137	1133	2270	1857	1857	3714	3454	3445	6899
25-29	355	347	702	978	1004	1982	1508	1447	2955	2841	2798	5639
30-34	304	278	582	805	840	1645	1085	1061	2146	2194	2179	4373
35-39	219	230	449	654	634	1288	866	855	1721	1739	1719	3458
40-44	210	192	402	559	515	1074	704	703	1407	1473	1410	2883
45-49	200	170	370	480	449	929	674	681	1355	1354	1300	2654
50-54	178	152	330	357	369	726	569	554	1123	1104	1075	2179
55-59	135	119	254	306	299	605	504	439	943	945	857	1802
60-64	93	99	192	196	244	440	339	336	675	628	679	1307
65-69	74	75	149	188	189	377	337	335	672	599	599	1198
70-74	37	43	80	128	135	263	224	197	421	389	375	764
75+	51	56	107	176	196	372	226	214	440	453	466	919
Total	4119	3971	8090	11020	10849	21869	17403	16741	34144	32542	31561	64103

### 1990 ALASKA NATIVE POPULATION BY ETHNICITY

AGE	<u>ALEUT</u>			<u>INDIAN</u>			<u>ESKIMO</u>			<u>TOTAL</u>		
	MALES	FEMALES	TOTAL	MALES	FEMALES	TOTAL	MALES	FEMALES	TOTAL	MALES	FEMALES	TOTAL
0-4	628	614	1242	1891	1933	3824	3503	3334	6837	6022	5881	11903
5-9	635	570	1205	1768	1701	3469	2751	2751	5502	5154	5022	10176
10-14	470	466	936	1539	1433	2972	2198	2104	4302	4207	4003	8210
15-19	392	373	765	1374	1232	2606	1880	1859	3739	3646	3464	7110
20-24	378	390	768	1196	1190	2386	1899	1843	3742	3473	3423	6896
25-29	495	481	976	1435	1424	2859	2160	2069	4229	4090	3974	8064
30-34	452	423	875	1396	1467	2863	1770	1880	3650	3618	3770	7388
35-39	377	376	753	1308	1231	2539	1494	1486	2980	3179	3093	6272
40-44	306	280	586	924	964	1888	1050	1020	2070	2280	2264	4544
45-49	242	233	475	766	703	1469	839	865	1704	1847	1801	3648
50-54	183	191	374	602	570	1172	629	637	1266	1414	1398	2812
55-59	183	166	349	498	475	973	608	659	1267	1289	1300	2589
60-64	126	120	246	332	388	720	479	504	983	937	1012	1949
65-69	115	117	232	270	279	549	394	415	809	779	811	1590
70-74	46	66	112	154	220	374	232	259	491	432	545	977
75+	60	98	158	259	323	582	357	473	830	676	894	1570
Total	5088	4964	10052	15712	15533	31245	22243	22158	44401	43043	42655	85698



## SERVICE UNIT AND TRIBAL HEALTH ORGANIZATIONS

### Anchorage Service Unit

Aleutian/Priblof Islands Association Incorporated  
 Copper River Native Association  
 Kenaitze Indian Tribe  
 Kodiak Area Native Association  
 Native Village of Eklutna  
 Native Village of Tyonek  
 Ninilchik Traditional Council  
 North Pacific Rim  
 Seldovia Village Tribe  
 South Kachemak, Incorporated, Alcohol Program  
 Southcentral Foundation  
 St. George Island Traditional Council

### Mt. Edgecumbe/Annette Island Service Unit

Ketchikan Health Clinic - IHS  
 Ketchikan Indian Corporation  
 Southeast Alaska Regional Health Corporation  
 Yak-Tat Kwaan Incorporated  
 Metlakatla Indian Community

### Barrow Service Unit

North Slope Borough Health and Social Services  
 Ukpeagvik Inupiat Corporation

### Bristol Bay Area Service Unit

Bristol Bay Area Health Corporation

### Interior Alaska Service Unit

Fairbanks Native Association  
 Tanana Chiefs Conference  
 Tanana IRA Native Council

### Kotzebue Service Unit

Maniilaq Association

### Norton Sound Service Unit

Norton Sound Health Corporation

### Yukon-Kuskokwim Delta Service Unit

Kuskokwim Native Association  
 Yukon-Kuskokwim Health Corporation

## 1980 Census Population for Alaska Natives by Service Unit

1.	Anchorage			17544
2.	Interior			6759
3.	Mt. Edgecumbe			9782
4.	Yukon-Kuskokwim	13953	}	17641
	Bristol Bay	3688		
5.	Barrow	2600	}	12321
	Kotzebue	4547		
	Norton Sound	5174		
	Total			<hr/> 64074

## ICD-O, Second Edition

Primary Site (Topography)	Code	Excluding Type
Oral Cavity and Pharynx:		
Salivary Gland	C079-C089	9590-9970
Nasopharynx	C110-C119	9590-9970
Gum & Other Oral Cavity	C030-C039;C050-C059;C060-C069	9590-9970
Other Oral Cavity and Pharynx	C000-C009;C019-C029;C040-C049 C090-C109;C129;C130-139 C140-C148	9590-9970 9590-9970 9590-9970
Digestive System:		
Esophagus	C150-C159	9590-9970
Stomach	C160-C169	9590-9970
Colon excluding rectum	C180-C189;C260	9590-9970
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Rectosigmoid Junction	C199	9590-9970
Liver	C220	9590-9970
Gallbladder	C239	9590-9970
Other Biliary	C240-249;C221	9590-9970
Pancreas	C250-C259	9590-9970
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Soft Tissue:	C380;C470-C479;C490-C499	9590-9970
Skin:		
Melanomas	C440-C449 (TYPES 872-879)	
Other non-Epithelial Skin	C440-C449	8000-8004 8010-8012 8070-8076 8090-8096 8720-8790 9700-9701
Breast:	C500-C509	9590-9970
Female Genital System:		
Cervix uteri	C530-C539	9590-9970
Corpus uteri/Uterus, NOS	C540-C549;C559	9590-9970
Ovary	C569	9590-9970
Other Female Genital Organs	C510-C519;C529;C570-C589	9590-9970
Male Genital:		
Prostate	C619	9590-9970
Testis	C620-C629	9590-9970
Other Male Genial Organs	C600-C609;C630-C639	9590-9970

Primary Site	Code	Excluding Type
Urinary System:		
Bladder	C670-C679	9590-9970
Kidney & Renal Pelvis	C649-C659	9590-9970
Other Urinary Organs	C669;C680-C689	9590-9970
Eye & Orbit:	C690-C699	9590-9970
Brain & Nervous System:		
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Lymphomas:		
Hodgkin's	TYPES 9650-9667	
Non-Hodgkin's	TYPES 9590-9595 TYPES 9670-9714	
Multiple Myeloma	TYPES 9731-9732: FOR ALL SITES	
Leukemia	TYPES 9800-9804;9820-9827;9830; 9840-9842;9850;9860-9864;9866- 9868;9870;9880;9890-9894;9900; 9910;9930-9931;9941	
Ill Defined & Unspecified:	TYPES 9720-9723;9740;9741;9760-9764;9950-9989 C760-C768;C809 C420-C424 C770-C779	9590-9970 9590-9940 9590-9750



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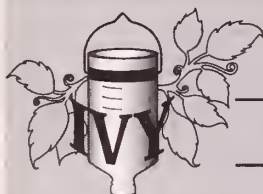
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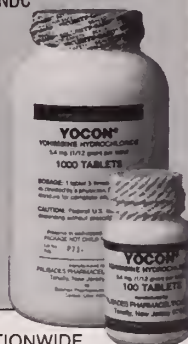
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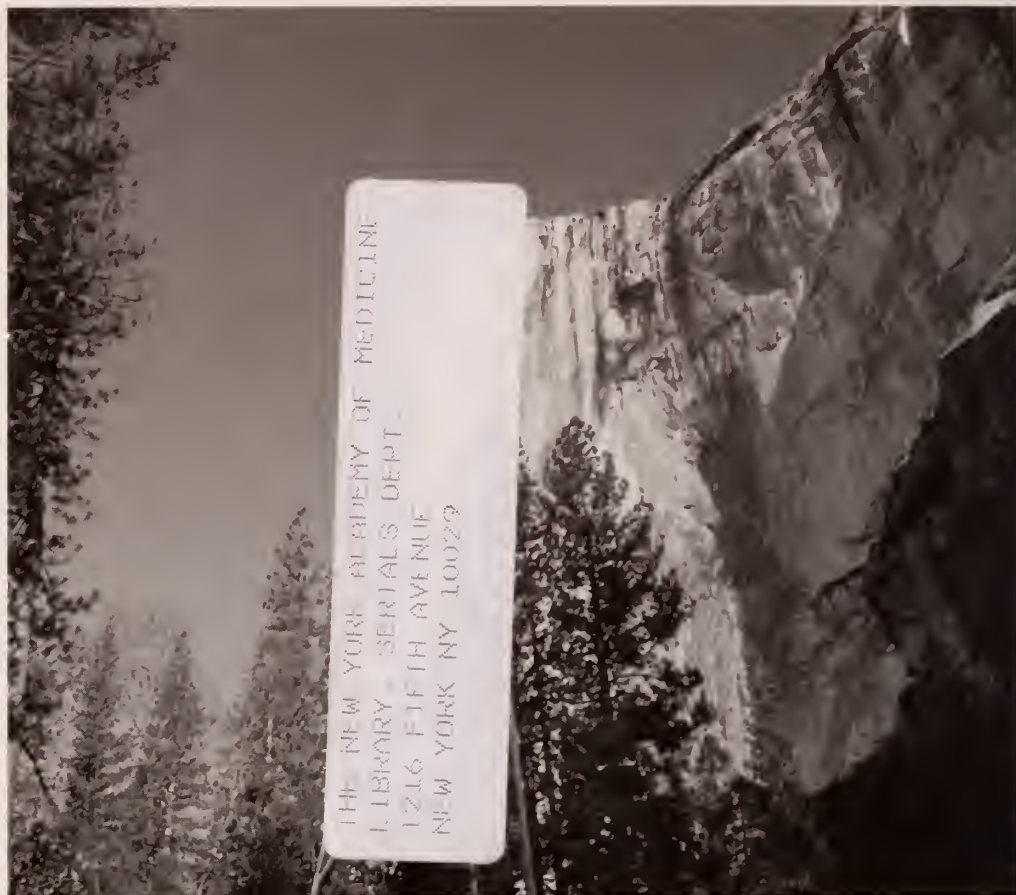
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# ALASKA MEDICINE

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# We Agree.

Every state medical society, 64 medical specialty societies, and the American Medical Association agree that any health system reform legislation must contain the principles outlined in the letter below:

February 23, 1994

Dear Senator/Representative;

As physician organizations, we agree on the need for health system reform legislation that gives every American universal coverage for health care and effectively controls rising health costs, while ensuring quality patient care. These principles have been articulated by numerous medical organizations in their various health system reform policies and proposals. They remain the foundation of our legislative agenda, which is to enact laws that assure universal coverage for a standard set of health benefits, regardless of employment or economic status.

We believe that any measure adopted by the Congress should:

- Achieve universal coverage through a program where responsibility is shared by employers, individuals, and government in paying for health care coverage.
- Assure that every American has his/her choice of health plans, physicians, and other providers.
- Establish competition in the marketplace as a method of slowing the rate of growth in health spending.
- Give patients price and quality information to permit them to make informed decisions.
- Eliminate needless bureaucracy to create an efficient, streamlined, and coordinated system that minimizes red tape for patients, physicians, and other providers. Furthermore, health system reform must leave medical decision-making in the hands of physicians and their patients.

We believe that to enable physicians to best serve the interests of their patients, meaningful health system reform also must contain these elements:

- Significant antitrust relief that enables physicians to have a strong voice to balance the growing corporate and government domination of health care.
- Allow for physician-directed health care networks.
- Enhanced self-regulatory powers that would enable the profession to effectively police itself and its members without the threat of unwarranted litigation.

We also believe that major reforms in the professional liability system must be enacted, including a \$250,000 cap on non-economic damages, limits on plaintiff attorneys' fees, and other measures that would minimize defensive medicine.

Every American will be affected by this legislation. The focus of policy-makers should be on how their decisions will affect patient care. Any system that raises significant barriers between patients and physicians will not provide the quality care our nation expects and deserves. We believe the above principles outline a framework for establishing constructive, effective, and needed health system reform.

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TABLETS

\* In studies with CLARITIN Tablets at doses 2 to 4 times higher than the recommended dose of 10 mg, a dose-related increase in the incidence of somnolence was observed.

† Relief began in 13% of treated patients vs 4% of placebo-treated patients within 30 minutes ( $P=.04$ ). At 2 hours, 48% of patients receiving placebo experienced relief. Distribution of onset times was significantly earlier for CLARITIN Tablets vs placebo ( $P=.03$ ).

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**BRIEF SUMMARY**  
(For full Prescribing Information see package insert)

**INDICATIONS AND USAGE**  
CLARITIN Tablets are indicated for the relief of nasal and non-nasal symptoms of seasonal allergic rhinitis.

**CONTRAINDICATIONS**  
CLARITIN Tablets are contraindicated in patients who are hypersensitive to this medication or to any of its ingredients.

**PRECAUTIONS**  
**General:** Patients with liver impairment should be given a lower initial dose (10 mg every other day) because they have reduced clearance of CLARITIN Tablets.

**Drug Interactions:** The coadministration of a single 20 mg dose of CLARITIN Tablets (double the recommended daily dose) and a 200 mg dose of ketoconazole twice daily to 12 subjects resulted in increased plasma concentrations of loratadine (180% increase in AUC) and its active metabolite, desloratadine (56% increase in AUC). However, no related changes were noted in the QTc on ECGs taken at 2, 6, and 24 hours after the coadministration of loratadine and ketoconazole. Also, there were no significant differences in clinical adverse events between CLARITIN Tablet groups with or without ketoconazole.

Other drugs known to inhibit hepatic metabolism should be coadministered with caution until definitive interaction studies can be completed. The number of subjects who concomitantly received macrolide antibiotics, cimetidine, ranitidine or theophylline along with CLARITIN Tablets in controlled clinical trials is too small to rule out possible drug-drug interactions. There does not appear to be an increase in adverse events in subjects who received oral contraceptives and CLARITIN Tablets compared to placebo.

**Carcinogenesis, Mutagenesis, and Impairment of Fertility:** In an 18-month oncogenicity study in mice and a 2-year study in rats, loratadine was administered in the diet at doses up to 40 mg/kg (mice) and 25 mg/kg (rats). In the carcinogenicity studies, pharmacokinetic assessments were carried out to determine animal exposure to the drug. AUC data demonstrated that the exposure of mice given 40 mg/kg of loratadine was 3.6 (loratadine) and 18 (active metabolite) times higher than a human given 10 mg/day. Exposure of rats given 25 mg/kg of loratadine was 28 (loratadine) and 67 (active metabolite) times higher than a human given 10 mg/day. Male mice given 40 mg/kg had a significantly higher incidence of hepatocellular tumors (combined adenomas and carcinomas) than concurrent controls. In rats, a significantly higher incidence of hepatocellular tumors (combined adenomas and carcinomas) was observed in males given 10 mg/kg and males and females given 25 mg/kg. The clinical significance of these findings during long-term use of CLARITIN Tablets is not known.

In mutagenicity studies, there was no evidence of mutagenic potential in reverse (AMES) or forward point mutation (CHO-HGPRT) assays, or in the assay for DNA damage (Rat Primary Hepatocyte Unscheduled DNA Assay) or in two assays for chromosomal alterations (Human Peripheral Blood Lymphocyte Clastogenesis Assay and the Mouse Bone Marrow Erythrocyte Micronucleus Assay). In the Mouse Lymphoma Assay, a positive finding occurred in the nonactivated but not the activated phase of the study.

Loratadine administration produced hepatic microsomal enzyme induction in the mouse at 40 mg/kg and rat at 25 mg/kg, but not at lower doses.

Decreased fertility in male rats, shown by lower female conception rates, occurred at approximately 64 mg/kg and was reversible with cessation of dosing. Loratadine had no effect on male or female fertility or reproduction in the rat at doses of approximately 24 mg/kg.

**Pregnancy Category B:** There was no evidence of animal teratogenicity in studies performed in rats and rabbits. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, CLARITIN Tablets should be used during pregnancy only if clearly needed.

**Nursing Mothers:** Loratadine and its metabolite, desloratadine, pass easily into breast milk and achieve concentrations that are equivalent to plasma levels with an AUC<sub>0-12</sub>/AUC<sub>0-12</sub> ratio of 1.17 and 0.85 for the parent and active metabolite, respectively. Following a single oral dose of 40 mg, a small amount of loratadine and metabolite was excreted into the breast milk (approximately 0.03% of 40 mg over 48 hours). A decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother. Caution should be exercised when CLARITIN Tablets are administered to a nursing woman.

**Pediatric Use:** Safety and effectiveness in children below the age of 12 years have not been established.

#### ADVERSE REACTIONS

Approximately 90,000 patients received CLARITIN Tablets 10 mg once daily in controlled and uncontrolled studies. Placebo-controlled clinical trials at the recommended dose of 10 mg once a day varied from 2 weeks to 6 months' duration. The rate of premature withdrawal from these trials was approximately 2% in both the treated and placebo groups.

REPORTED ADVERSE EVENTS WITH AN INCIDENCE OF MORE THAN 2% IN  
PLACEBO-CONTROLLED ALLERGIC RHINITIS CLINICAL TRIALS  
PERCENT OF PATIENTS REPORTING

	LORATADINE 10 mg QD n = 1926	PLACEBO n = 2545	CLEMASTINE 1 mg BID n = 536	TERFENADINE 60 mg BID n = 684
Headache	12	11	8	8
Somnolence	8	6	22	9
Fatigue	4	3	10	2
Dry Mouth	3	2	4	3

Adverse event rates did not appear to differ significantly based on age, sex, or race, although the number of non-white subjects was relatively small.

In addition to those adverse events reported above, the following adverse events have been reported in 2% or fewer patients:  
**Autonomic Nervous System:** Altered salivation, increased sweating, altered lacrimation, hyposensitivity, impotence, thirst, flushing.  
**Body As A Whole:** Conjunctivitis, blurred vision, earache, eye pain, lumbago, asthenia, weight gain, back pain, leg cramps, malaise, chest pain, rigors, fever, aggravated allergy, upper respiratory infection, angioneurotic edema.  
**Cardiovascular System:** Hypotension, hypertension, palpitations, syncope, tachycardia.  
**Central and Peripheral Nervous System:** Hyperkinesia, blepharospasm, paresthesia, dizziness, migraine, tremor, vertigo, dysphonia.  
**Gastrointestinal System:** Abdominal distress, nausea, vomiting, flatulence, gastritis, constipation, diarrhea, altered taste, increased appetite, anorexia, dyspepsia, stomatitis, toothache.  
**Musculoskeletal System:** Arthralgia, myalgia.  
**Psychiatric:** Anxiety, depression, agitation, insomnia, paranoia, amnesia, impaired concentration, confusion, decreased libido, nervousness.  
**Reproductive System:** Breast pain, menorrhagia, dysmenorrhea, vaginitis.  
**Respiratory System:** Nasal dryness, epistaxis, pharyngitis, dyspnea, nasal congestion, coughing, rhinitis, hemoptysis, sinusitis, sneezing, bronchospasm, bronchitis, laryngitis.  
**Skin and Appendages:** Dermatitis, dry hair, dry skin, urticaria, rash, pruritus, photosensitivity reaction, purpura.  
**Urinary System:** Urinary discoloration, altered micturition.

In addition, the following spontaneous adverse events have been reported rarely during the marketing of loratadine: peripheral edema, abnormal hepatic function, including jaundice, hepatitis, and hepatic necrosis; alopecia; seizures; breast enlargement; erythema multiforme; and anaphylaxis.

#### OVERDOSAGE

Somnolence, tachycardia, and headache have been reported with overdoses greater than 10 mg (40 to 180 mg). In the event of overdosage, general symptomatic and supportive measures should be instituted promptly and maintained for as long as necessary.

Treatment of overdosage would reasonably consist of emesis (ipecac syrup), except in patients with impaired consciousness, followed by the administration of activated charcoal to absorb any remaining drug. If vomiting is unsuccessful, or contraindicated, gastric lavage should be performed with normal saline. Saline cathartics may also be of value for rapid dilution of bowel contents. Loratadine is not eliminated by hemodialysis. It is not known if loratadine is eliminated by peritoneal dialysis.

Oral LD<sub>50</sub> values for loratadine were greater than 5000 mg/kg in rats and mice. Doses as high as 10 times the recommended clinical doses showed no effects in rats, mice, and monkeys.

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#### Reference

1. Bédard P-M, Del Carpio J, Drouin MA, et al. Onset of action of loratadine and placebo and other efficacy variables in patients with seasonal allergic rhinitis. *Clin Ther* 1992;14:268-275.

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# YOCON® YOHIMBINE HCl

**Description:** Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubiaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

**Action:** Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalamic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it, however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

**Indications:** Yocon® is indicated as a sympathicolytic and mydriatic. It may have activity as an aphrodisiac.

**Contraindications:** Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

**Warning:** Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

**Adverse Reactions:** Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.<sup>1,2</sup> Also dizziness, headache, skin flushing reported when used orally.<sup>1,3</sup>

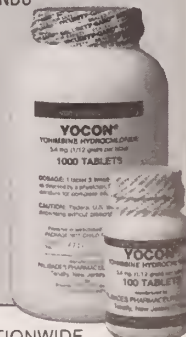
**Dosage and Administration:** Experimental dosage reported in treatment of erectile impotence.<sup>1,3,4</sup> 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to 1/2 tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.<sup>3</sup>

**How Supplied:** Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

#### References:

1. A. Morales et al., New England Journal of Medicine: 1221, November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical Letter, 27:2, July 4, 1983.
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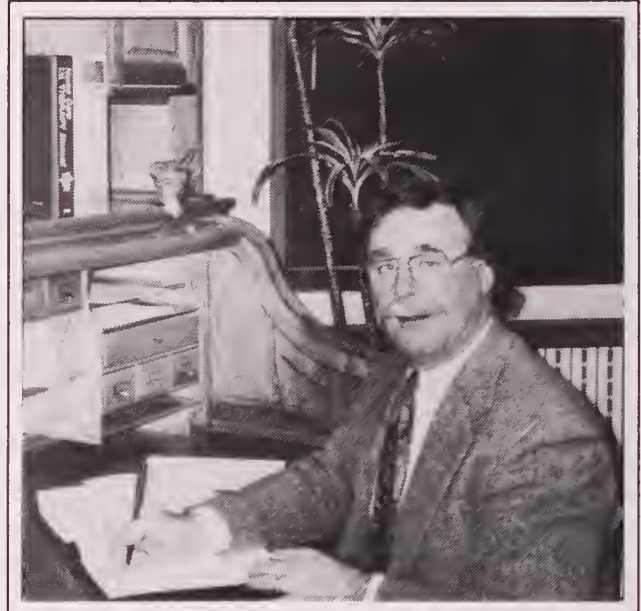




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About the cover: Children manning a blanket toss during Whale Feast  
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# Causes of Death of Residents of Anchorage Dying Elsewhere, 1985-1988

Rodman Wilson, M.D.<sup>(1)</sup>

## ABSTRACT

Three hundred fifty-five residents of Anchorage, Alaska, died away from their home city during the years 1985-1988. Typically, they were Caucasian, male, and in the middle years of life. Half died violently.

## INTRODUCTION

In the years 1985-88, 3,435 persons died within the boundaries of Anchorage, Alaska. Of these 2,529 dwelled in Anchorage (1-4).

Three hundred fifty-five other residents of Anchorage are known to have succumbed away from the city during the same period. Of these, 192 died elsewhere in Alaska and 163\* in other states or Canada (Table 1).

## METHOD

The Alaska Bureau of Vital Statistics furnished copies of certificates of death of residents of Anchorage dying in other locations in Alaska and copies of death certificates or equivalent notice from other states and Canada. States routinely notify the home state when a non-resident dies within their borders, but sometimes communication fails. Other nations do not notify regularly, but Canada does.

Table 1

Residents of Anchorage Dying Elsewhere  
1985-1988

	Within Alaska	Out-of-State	Total
1985	49	50	99
1986	55	33	88
1987	40	33	73
1988	48	47	95
Total	192	163	355

\* comprising 30% of 551 Alaskans known to have died out-of-state during the quadrennium (5)

## FINDINGS

Causes of death are listed in Tables 2 and 3.

Overall, of the 355 individuals who died away from Anchorage, 174 (49%) died from violence, 175 (49%) from illness and 6 (2%) from unstated causes.

Table 2

Causes of Death of Residents of Anchorage  
Dying Elsewhere in Alaska, 1985-1988

Violent Deaths		
aircraft crash	42	
motor vehicle collision	39	
drowning	26	
homicide	10	
suicide	8	
mountain climbing	7	
drug overdose, including alcohol	5	
gunfire, unintentional	2	
disappeared on land	2	
disappeared on water	2	
avalanche	1	
tree fell on head	1	
explosion on boat	1	
carbon monoxide from cabin stove	1	
fire	1	
hit on head on job	1	
subtotal	149	(78%)
Nonviolent Deaths		
atherosclerotic heart disease	26	
heart disease, other	4	
pneumonia	3	
cancer	2	
aneurysm of aorta, ruptured	2	
sudden infant death syndrome	2	
diabetes mellitus	1	
gastroenteritis	1	
sepsis	1	
old age	1	
subtotal	43	(22%)
total	192	(100%)

<sup>(1)</sup>800 M Street, Anchorage, AK 99501

Three-quarters (78%) of residents of Anchorage dying away from home but within Alaska died violently, most commonly in aircraft and highway crashes or by drowning. Of those not dying violently (22%), by far the most died from atherosclerotic heart disease (Table 2).

On the other hand, only one-seventh (15%) of 163 Anchorage residents who perished out-of-state died of injury, most often in motor vehicle crashes. Except in a few instances (4%) where cause of death was not listed on documents received from other jurisdictions, the rest fell to disease: 32% from cardiovascular disorders, 25% from cancer, 12% from congenital defects, and 12% from a variety of other illnesses (Table 3).

Notice that cirrhosis of the liver is classified as an illness, rather than as violence (poisoning), because there

was no mention of alcoholism on the certificates of deaths from other states. In previous studies (1-4), there was opportunity to query attending physicians or pathologists about the role of alcohol in such cases.

The difference in proportions of violent deaths among residents of Anchorage dying at home (21%) (4) or elsewhere (49%) is statistically highly significant ( $P<0.005$ ), but the difference between those dying violently at home (21%) or out-of-state (15%) could be due to chance ( $P=0.08$ ).

Residents dying at home during the years 1985-88 were more often male (58%) than female (42%) in a city 51% male (6). When residents died away from home, 74% were male. The difference is significant ( $P<0.005$ ). The spread was even more pronounced when residents died elsewhere in Alaska: 83% of these were male. Among residents succumbing out-of-state, 64% were male—not a significant difference from the proportion of males (58%) dying at home ( $P>0.10$ ).

Eighty-eight percent of residents dying away from home were Caucasian, 5% Native American, 3% Black, 3% Asian, and 1% of other or unknown race. Among Anchorage residents dying at home 81% were Caucasian—a significant difference ( $P<0.005$ ).

Overall, median age at death was 41 years. For those dying within Alaska but away from Anchorage, median age at death was 38 years and for those dying out-of-state 51 years. Mean ages at death, excluding those dying before age 1, for the whole group and the two subcategories were, respectively, 46.1, 40.9, and 52.9 years. These ages all considerably less than the median age of 60 years and the mean age of 57.8 years of people dwelling in Anchorage and expiring there.

Residents met death within Alaska at a great number of sites both urban and rural (Table 4). Residents died outside Alaska in 28 states, most often in Washington, California, Arizona, and Minnesota (Table 5). Six died in Canada.

## COMMENT

The 355 residents of Anchorage who died away from their home city represent 12% of the 2,884 inhabitants who died in the four-year period, 1985-88. Every eighth resident who died during this interval expired out-of-town. Fifty-four percent perished elsewhere in Alaska; 46 percent died out-of-state.

Death by violent means was common among citizens of Anchorage during these years (4), but it was even more frequent as a cause of demise away from home, especially if death occurred elsewhere in Alaska. Many of these deaths happened, clearly or putatively, during recreational activities such as flying, driving, boating, and mountaineering.

Residents expiring out-of-state were older than

Table 3

### Causes of Death of Residents of Anchorage Dying Outside Alaska, 1985-1988

#### Violent Deaths

motor vehicle collision	12	
aircraft crash	4	
homicide	4	
suicide	3	
drowning	1	
drug overdose, narcotic	1	
subtotal	25	(15%)

#### Non-violent Deaths

cancer	41	
atherosclerotic heart disease	22	
prematurity and congenital anomalies	20	
heart disease, not atherosclerotic or congenital	10	
aortic or iliac aneurysm	6	
cerebral hemorrhage	4	
cerebral thrombosis	4	
chronic obstructive pulmonary disease	4	
cirrhosis of liver	4	
pulmonary embolism	3	
generalized atherosclerosis	3	
Alzheimer's disease	2	
pneumonia	2	
end-stage renal disease	2	
miscellaneous causes	5	
subtotal	132	(81%)
unstated or unknown cause	6	(4%)
total	163	(100%)

Table 4

Place of Death of Residents of Anchorage  
Dying Elsewhere in Alaska, 1985-1988

Palmer	11	Bethel	2
Cook Inlet	10	Cantwell	2
Parks Highway	8	Chakachamna	2
Sterling Highway	8	Galena	2
Soldotna	7	Hatcher Pass	2
Homer	7	Heart Lake	2
Fairbanks	7	Houston	2
Juneau	7	Kachemak Bay	2
at sea	5	Kenai	2
Glenn Highway	5	Ketchikan	2
Seward Highway	4	Mount Foraker	2
Point McKenzie	4	Mount McKinley	2
Burns Glacier	3	Mount Torbert	2
Gulkana	3	Sterling	2
Lake Clark	3	Talkeetna	2
Prudhoe Bay	3	Yentna River	2
Seward	3	other locations, 1 ea	53
Valdez	3		
Wasilla	3		
Willow	3	total	192

Anchoragites meeting death elsewhere in Alaska (but not so old as residents dying in Anchorage) and much more likely to be afflicted with disorders like cancer, cardiovascular disease, and lethal congenital defects. Many had undoubtedly gone to cities such as Seattle, San Francisco and Rochester, Minnesota for medical care.

## SUMMARY

Three hundred fifty-five residents of Anchorage, Alaska are known to have died away from their home city during the years 1985-88. This was 12% of all known deaths of residents during this quadrennium. When citizens of Anchorage died away from home, they were typically Caucasian, male, in the middle years of life, and prone to die violently.

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Table 5

Place of Death of Residents of Anchorage  
Dying Outside Alaska, 1985-1988

WA	47	NY	4
Seattle	37	OR	4
CA	25	YT, Canada	4
LA area	8	FL	3
SF area	7	KS	3
AZ	13	AL, Canada	2
MN	10	ID	2
Rochester	6	IL	2
TX	8	NB	2
UT	7	WI	2
HI	6	other states, 1 ea	11
CO	4		
NV	4	total	163



# Systemic Lupus Erythematosus and Systemic Scleroderma in Patients from the Aboriginal People and the Newcomers of Yakutia Under the Extreme Conditions of the Far North

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## INTRODUCTION

Systemic lupus erythematosus (SLE) and systemic scleroderma (SSD), being the leading forms of systemic diseases from the large collagenosis group are characterized by a considerable degree connected with a number of environmental factors, revealing the first signs of a disease, or having influence on frequency and gravity of relapses.

In literature there is some information on the clinical SLE and SSD peculiarities depending not only on living in locations of climato-geographical zones (12,14,17,24,29,36), but also on ethnic factors (1,3-5,7,9,25,28,30,35).

The systemic diseases peculiarities in the regions with cold climate practically without any factual informations on SLE and SSD under the Far North conditions have been described in a few papers (10,11,18,21,23).

Not only cold, but sharply continental climate in Yakutia (located in the North-East of Asian part of Russia), is characterized by unusual, extreme living conditions. The atmospheric winter temperature is -55° - 62° C below zero, and in summer is +35° - 40° C. That fact distinguishes the territory of the republic as the region with harsh continental climate as compared with the other populated areas of the North.

High level of sun radiation, which is typical of the southern republics of CIS (particularly in Kazakhstan) is very substantial. It means that this high insolation level determines the high risk factor of the sun radiation during the polar day.

Thus, sharply expressed overfall of natural ultraviolet radiation during a year is defined by its full or almost absence in winter months at substantial lowering in autumn and in early spring. But at the same time, the amount of natural UV-radiation exceeds the daily

physiological need of organism at 24 times in short but hot summer (2,31).

The richness of this region by minerals, including deposits of gold, diamonds, natural gas, rare metals and others, determines the intensive development of the mining industry and most of its factors, such as dust of silicium dioxide, vibration, toxic and irritative components, aggravates the risk of the systemic diseases development, including large collagenosis. Thus, systemic diseases with professional pathology are the result of silicoarthritis, silicolupus and silicoscleroderma (6,8,13,15,16,19,20, 26,27,32,34).

We were greatly interested in analyzing the course of the most widespread systemic diseases in ethnically different groups of population, including the aboriginal people of the North. The expressed deflection on the side of enzyme system, connected with the connective tissue formation in particular are often occurred in the aboriginal people. This fact reveals more often congenital defects of the brachial tree, ventricular septum defects and atrial septum defects at the patients of this region and relatively greater frequency of rheumatoid arthritis and Marfan's syndrome (0.6 versus 0.2 in population) (22).

## METHODS

Clinical manifestations of SLE and SSD have been studied at 79 patients (59 SLE patients and 20 SSD) taking into consideration ethnic factors. Patients were examined in specialized department of the Republican Medical Center and the clinical city hospital in Yakutsk. Specialists in narrow fields of medicine took part in deep clinical testing. ECG, echocardiographic of the heart, x-ray examination of the chest organs, joints, the ultrasound investigation of inner organs, routine laboratory studies and special tests, general blood analysis, general urine analysis, urinary sediment examination, biochemical examination of the liver and kidneys function: alanine transaminase, asparagine transaminase, bilirubin, blood protein and correlation of its fraction, blood urea and

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blood creatinine were made. LE-cells, immunoglobulins A, M, G were also studied. All the patients with SLE correspond to not less than four criteria of ARA 1982 (33).

The data have been analyzed by the method of varying statistics using Student's criteria (the results were considered to be reliable by  $P < 0.05$ ).

## RESULTS

The data obtained on clinical manifestations of SLE and SSD 79 patients have been summarized. Out of 79 patients 59, patients suffered from SLE and 20 from SSD.

The number of the aboriginal people of Yakutia was equal to 47 patients and 32 patients (Russian and Ukrainian) migrants of the first generation (see Table 1).

SLE spreading among the Yakuts was 1.55 per 10,000 men, among the Europeans - 0.48 per 10,000 ( $P < 0.05$ ).

The number of SLE and SSD patients among the aboriginal people of the North was equal to 59.5 percent as compared to all the studied cases. Taking into consideration the fact that the number of the aboriginal people is equal to one-third of the whole population of Yakutia, we can state that systemic diseases occurred more often among the Yakuts, particularly SLE (64.4 percent) ( $P < 0.05$ ).

The specific professional occupations of newcomers (miners of vibrodangerous professions, contacting with the dust of silicium dioxide, the personal, connected with hydrocarbon compounds are of special interest. In four cases, the migrants of the first generation had had some symptoms of systemic diseases before they came to the North. They had the following manifestations of the disease: Reino's syndrome, pain in the joints, dermatitis; but they were not dangerous. Under the extreme

conditions of the North the disease developed quickly in distinctly expressed forms.

Agriculture workers, a hunter, employees were patients with SLE and SSD among the aboriginal people.

The results of the clinical manifestations of each nosological form are as follows.

## SLE

Fifty-nine cases were studied in dynamics: 38 were aboriginal patients and 21 migrants. Women prevailed in both ethnic groups, while among the Yakuts the correlation was 1.0:9.0 and among migrants - Europeans - 1.0:10.5. The onset of the disease at the overwhelming majority constituted 83 percent as to young age (see Table 2). At three patients SLE had begun before they came to the North and developed quickly during the following years.

According to the data obtained, the symptoms of the disease in both ethnic groups were similar, having a subacute and chronic character with frequent relapses (see Table 2). The skin, joints, kidneys impairment were the main symptoms of the disease during a number of years.

At the same time, severe forms of visceral lungs and kidney impairment, more frequent generalization of the process with polyserositis and involvement of central nervous system have been noted among the aboriginal people.

Skin impairment in the form of erythema on the type of "butterfly" were noted at the majority of examined patients, sometimes coinciding with the early spring sun radiation, being as a provocation moment (see Table 3). In two cases lupus dermatitis had an unusual localization, while specific outlines of "butterfly" were on the thorax

Table 1							
Patients Distribution According to Age, Sex and Nationality							
Nationality		Age	15-20	21-30	31-40	41-50	51+
		Sex					
SLE	Yakuts	F	5	12	15	3	1
		M	-	2	-	-	-
	Russian and Ukrainian	F	5	3	6	3	2
		M	-	-	1	1	-
SSD	Yakuts	F	-	4	1	2	2
		M	-	-	-	-	-
	Russians and Ukrainians	F	1	2	2	-	-
		M	-	1	3	2	-

Table 2

## SLE Patients Distribution According to the Course of the Disease and the Activity Degree

		Process Form				Activity Degree					
Nationality	Sex	Accute	%	Subacute	%	Chronic	%	I	%	II	%
Yakuts	F	1		21		14		14		15	
			2.6		60.5		36.9		36.9		42.1
	M	-		2		-		-		1	
										1	
Russians and Ukrainians	F	2		9		8		6		9	
			9.5		47.6		42.9		33.3		47.6
	M	-		1		1		1		1	
										-	

and, even on the small of the back. There were some other variants of lupus dermatitis with the hair loss and tropical disturbances.

The prognosis was often determined by kidneys impairment and had the form of lupus glomerulonephritis with nephrotic syndrome.

The symptoms, which were typical of systemic diseases, mainly collagenosis (the so-called "overlap-syndrome") were noted almost at 1/3 cases (31.6 percent) in aboriginal people of the North and only in two (9.5 percent) migrants of European origin.

Thus, in two cases, the onset of the disease manifested

in symptoms resembling dermatomyositis: paraorbitalis edema, myositis with the development of almost total movelessness. However, the further course of the disease with typical erythema on the face in the form of "butterfly," typical polyserositis and lupus pneumonitis determined the diagnosis of SLE. In two cases, the course of SLE in its onset manifestations was correlated not only with some signs of dermatomyositis, but with SSD as well.

The SLE variant similar to rheumatoid arthritis with the typical deformation of radio-carpal and intraphalangeal joints, however; with clinically short skin and visceral

Table 3

## The Number of Cases and Signs of Impairments on SLE in Two Ethnic Groups

Signs	Yakuts the number of cases		Russians and Ukrainians the number of cases	
	n	%	n	%
skin impairment	34	87.0	21	100.0
joints impairment	39	100.0	15	71.4
lungs	31	79.5	11	52.4
pleuritis	11	28.2	3	14.3
pericarditis	5	12.8	1	4.8
myocarditis	27	69.2	14	66.7
endocarditis Libman-Sacks	1	2.6	2	9.5
kidneys	32	82.0	13	61.9
liver	24	61.5	10	47.6
fever	26	66.7	15	71.4
blood	36	92.3	18	85.7
central nervous system	14	35.9	5	23.8
myositis	11	28.2	5	23.8
LE-cells	28	71.8	16	76.2



manifestations of SLE also occurred frequently among the aboriginal people of the North, as compared to the migrants.

Among the aboriginal patients, 76.3 percent had the impairment of large and middle joints, including shoulder and pelvifemur joints along with the small joints impairment with passing arthralgi pains in joints. And in European patients the number is equal to 38 percent.

Intensive therapy defined a prolonged relapse in 1/3 of the cases in both ethnic groups. The lethal outcomes taken place in 6 cases (3 cases in Yakuts and 3 in Russians) were due to the kidneys impairment, complicated by the specific infection (generalized tuberculosis) and sepsis.

## SSD

Twenty persons, 11 Europeans and nine Yakuts, had SSD. Relatively greater frequency of nontypical cases of the course of the disease, four out of 20, is of special interest. Thus, in three patients the "debut" of the disease was characterised by the lung impairment with Reino's syndrome manifestation, typical skin syndromes and other specific features of the disease three to five years later. In one of the cases the disease had been interpreted as pneumosclerosis of tuberculosis ethiology for a long period of time. The lung impairment defined the lethal outcome with hystological confirmation of sclerodermic lung in one case. In the second case, relatively early diagnosis of sclerodermic lung and rationally done therapy were the result of the relapse with relatively mild course of the disease.

In five cases, SSD proceeded with heart impairment as the main syndrome. At the same time in one of the cases, in Yakutian girl of 21, the heart impairment proceeded without any other manifestations of the disease (the so-called "scleroderma without derma") and that was the reason for the wrong diagnosis of rheumatic valvular heart disease.

The lethal outcome, stipulated by decompensation with the following hystological investigation allowed us to define the diagnosis and reveal the typical sclerodermic heart with vast fibrosing myocard.

Only in one of the cases of SSD the Yakutian woman patient of 63 had a relatively mild form. The disease was characterised by the face skin impairment, the presence of Reino's syndrome, sclerodactilia, calcinatis later with relatively moderately expressed lung processes. At the age of 66 the patient died of cerebral effusion due to hypertonic disease.

And more frequent nontypical manifestations of gastrointestinal tract impairment should be considered as some peculiarities of sclerodermic in the North. So, the erosion of the lower one-third of esophagus were revealed in one-third cases and in one case leukoplakia of

the same localization was revealed. In one of the cases the 38 year-old Russian patient had Reino's syndrome before he came to the North. The disease progressed quickly because of the patient contact cooling vibration. The galloping course of the disease with the skin, lung impairment on the type of fibrosing alveolitis, the heart impairment up to the occurrence of pseudoinfarction changes was characterised by the changes on the side of gastrointestinal tract. Despite the intensive therapy (corticosteroids, cytostatics, D-penicillamin, plasmaferes), episodes of dynamic intestinal obstruction were complicated by total necrosis of the vast parts of small and large intestines, which became the reason for special operative intervention with the lethal outcome during the past period.

## DISCUSSION

According to the data obtained the frequency of the two main systemic diseases in the group of large collagenosis - SLE and SSD is high among the Northern population: 1.55 percent among the Yakuts and 0.48 percent among the Europeans. This speaks of the presence of a large complex of provoking factors.

More higher level of SLE spreading among the Yakutian population, as compared with migrants from the European part of Russia may be determined by genetic factors, including the tendency of disturbing the collagen metabolism, which speaks of more spreading of Marfans's syndrome (0.6 versus 0.2 in population) and rheumatoid arthritis among the aboriginal people of Yakutia. Among the patients from the newcomers the dependence on the industrial factors contact, serious attitude towards the prognosis in the cases of ill-time disease diagnostics, particularly among the migrants from the middle-climate zone to the North, should be of special attention. It should be considered that greater frequency of visceral lesions, including the cases without obvious classical manifestations on the side of the skin, ("lupus sine lupi" and "scleroderma without derma") and also substantial specific gravity, mostly among the aboriginal people of the North, the cases of overlap-syndrome, which sometimes interpreted as the combination of two or more forms of pathology, or as "indifferentiated collagenosis."

Relatively smaller involvement of the skin integument in the patients of Yakutian nationality with serious visceral and sometimes showing the impairment prognosis may be connected with darker color of the skin and integument of the defensive role of melanin. Negroes and Orient people have the same peculiarities.

It should be noted that relatively high level of disabled persons is equal to 42 percent among the SLE patients of Yakutian nationality and, 47.6 percent among the migrant Europeans.

## SUMMARY

There is some information on the course of two main forms of large collagenoses - systemic lupus erythematosus (SLE) and systemic scleroderma (SSD) under the conditions of the North of the Asian part of Russia (Yakutia) in this paper. Seventy-nine cases (59 SLE patients and 20 SSD patients) belonging to different ethnic groups were studied. There were 47 patients of Yakutian nationality, among them SLE 38 patients and SSD nine patients. There were 32 patients (SLE - 21, and SSD - 11) migrant Europeans.

It has been proved that the aboriginal people of the North are more subject to SLE and SSD diseases as compared to the migrants. It has also been proved that the greater spreading of the diseases, with the collagen metabolism disturbance in the first ethnic group, including Marfan's syndrome and rheumatoid arthritis may be explained by genetic peculiarities. Some ethnically stipulated differences in clinical manifestations of two large collagenosis were revealed. Thus, during SLE smaller frequency of the skin impairment in the Yakuts (due to natural hyperpigmentations) is connected with the considerable frequency of large joints impairment and more frequent course of SLE similar to rheumatoid variant with typical wrist deformation. One-third SLE and SSD patients of Yakut nationality reveal "overlap-syndrome", which are typical of other collagenoses, the so-called overlap-syndrome.

Some industrial factors (dust of silicon dioxide, vibration, hydrocarbon compounds) are the main reason for the diseases among the newcomers migrants of Russian and Ukrainian nationality. One case of silico-lupus was revealed.

Relatively high specific gravity of hard visceral impairment, which reveals the prognosis (sclerodermic lung, sclerodermic heart and kidneys, lupus visceritis) has been noted in both ethnic groups. Thus, in some cases, in persons of Yakutian nationality visceral pathology was not only "debut", but almost the only manifestation of the disease up to the end.

Clinical peculiarities of SLE and SSD under the extreme conditions of the North confirm their great role in the genesis of these forms of pathology processes adaptations disturbance and the immune overstrain and constitutional (genetic) factors as well.

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(continued on page 126)



# Marijuana: A Research Overview

Linda Rinaldi

## INTRODUCTION

Marijuana use is a subject surrounded by controversy. As one modern researcher notes, the controversy itself serves to cloud the issue. "The effects of marijuana on health in general have been marked by polarities of belief or interpretation of evidence often due to the particular prejudices of investigators" (Hollister 1988, 3). Only recently has the technology been available to adequately begin to unravel the biochemistry of this substance. Even though there is a substantial body of published literature, from opinions to studies, the complex pharmacology and toxicology of marijuana is still not completely understood.

The level of controversy, complexity of marijuana as a substance and the difficulty of research are not the only factors which cloud understanding. Another aspect important in considering abuse of marijuana is an understanding of dependent behavior and reasons for using marijuana. Human motivations and behavior are profoundly difficult to analyze and resistant to laboratory experimentation.

This paper cannot hope to examine all of the intricacies of this subject. It will, instead, begin with an overview of marijuana, its forms of administration, and some of the known chemicals and reactions involved. This will be followed by a discussion of the effects, both immediate and long-term, as well as potential therapeutic uses. Finally, the discussion will turn to a brief overview of marijuana abuse treatment and prevention.

A large volume of research and opinions have been assembled, as discussed above (also Kalant 1982). This paper relies heavily on *Marijuana and Health*, a report prepared by a Committee of the Institute of Medicine of the National Academy of Sciences. Published in 1982, it reviewed scientific information on marijuana and health published from 1975 on. Research subsequent to the 1982 report has likewise been substantial. This paper includes a selection of those reports as well.

## CHEMICAL AND PHYSICAL PROPERTIES OF MARIJUANA

Marijuana is a term commonly used to refer to *Cannabis sativa*. The plant is found in many places around the world, and has been used for its psychoactive effects and reputed medicinal qualities for centuries. It is usually

smoked or eaten and is known to produce an intoxicated state, or "high." Hashish is a resin collected from the plant and contains a higher concentration of psychoactive ingredients. Sinsemilla, which literally means "without seeds," is a form of marijuana cultivated for this quality and higher potency.

*Cannabis sativa* contains hundreds of chemicals. Only 61 of these are unique to the marijuana plant and have been termed "cannabinoids." Although research has been conducted for years, it was not until 1964 that the primary psychoactive ingredient was identified as delta-9-tetrahydrocannabinol or delta-9-THC, hereafter referred to as THC. The other key cannabinoids, delta-8-THC, cannabidiol, and cannabinol, have been studied to a much lesser extent.

When smoked, THC, other cannabinoids, chemicals and impurities present in marijuana pass through the lungs, into the blood stream, and then into body tissues. Eventually the substances are metabolized in cells, primarily liver cells, and pass out of the body. When eaten, THC passes through the digestive system and liver first, which clears much of the THC prior to circulation, although many of the metabolites remain psychoactive (Morgan, 1988).

THC and other cannabinoids are fat soluble. Metabolites resulting from conversion in the liver and elsewhere are more water soluble. The level of THC in the blood stream falls off fairly rapidly, as quickly as 30 minutes after first smoked. The level then declines at a much slower rate as metabolites of THC come back into the blood stream from the tissues in which they are stored. The level of THC in the blood and THC metabolites in the urine decline within a few hours of smoking, although metabolites can be detected in the urine for long periods following ingestion or smoking (Morgan, 1988).

This slow rate of decline is referred to as half-life, or the amount of time it takes to eliminate one-half of a quantity and then one-half of the remainder, etc. Estimates of this duration vary, although it can take a very long time for the urine to be completely clear, even several weeks. The effect of these metabolites and rapidity of metabolism are not fully understood, which contributes to the dispute over the effects of the drug. Some argue that the effect of residual THC in the tissue is substantial. On the other hand, "... [t]here may be a fairly rapid and complete metabolism of free delta-9-THC followed by slow release and metabolism of sequestered

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delta-9-THC and retained metabolites" (NAS 1982,23).

Another point confounding research is that potency of marijuana varies considerably, making dosages hard to determine. Individual smoking behavior varies substantially from person to person as well. Use of THC alone allows more control of dosage, but different inhalation patterns present experimental obstacles (Wu 1988, Perez-Reyes 1990). THC is often administered orally or injected in experiments, methods also raising some objections. The conclusion is that there is no easy way to research marijuana.

Several recent investigations have shown that specific receptors in the brain account for many of the perceived effects of marijuana. In the early 1980s, researchers (Martin 1988, Johnson 1988) were able to determine that the most psychoactive ingredient (delta-9-THC) did affect the cell, but that the effect was rapid and reversible with no permanent change, and that only certain cell types were involved.

Using synthetic cannabinoids and radioactive labeling, current research (Herkenham 1990) has verified the existence of those receptors in the brain with the highest concentrations in parts of the basal ganglia, hippocampus, cerebral cortex and cerebellum. Other substances did not bind to the receptor sites suggesting specificity to cannabinoids. The sites are consistent with characteristic effects of marijuana including short-term memory impairment, movement, and analgesic effects. Low concentrations of the receptors in the brain stem, which controls the heart and breathing, explain the absence of fatalities in humans associated with an overdose of cannabis.

More recent research was reported widely in newspapers in August, 1990 (Maugh 1990) and revealed the discovery of the receptor itself. This identification should eventually lead to the isolation of the naturally occurring correlate in the human body. This likewise shows promise for development of the synthetic substance, free of the side effects which have inhibited use in the past; and could be used in treatment of those who are marijuana dependent as well as other therapeutic applications.

## PREVALENCE AND USE

Two surveys sponsored by the National Institute of Drug Abuse have provided information on marijuana and other drug use for several years. The National Household Survey was initiated in 1971 and has been conducted on an annual or biannual basis since. Results are grouped by the age of the respondents into three categories — youth (12-17), young adults (18-25) and older adults (26 and older). The second survey, Monitoring the Future, focuses on high school seniors and has been conducted annually since 1975.

Use of marijuana rose steadily in the 20 years from 1960 to 1980, so that by that time almost 70 percent of the

young adults surveyed reported having tried marijuana at least once in their lives (NAS 1982, 36). Overall use of marijuana, as indicated by the annual survey of seniors, has declined over the past few years. While 60 percent of 1980 seniors reported having tried marijuana at least once, the level fell to about 50 percent in 1986, and continues to decline.

Attitudes about legalization of marijuana have also changed in recent years (Sommer, 1988). Students surveyed over several years are currently reporting a decreased number favoring legalization, which represents a steady decline since 1973. These findings agree with the national surveys which show a similar decline in acceptance.

Use in the general population is also declining, across all age groups. The number of people using marijuana once a week or more declined by 28 percent from 1985 to 1988. However, a reported increase in the availability of marijuana and a relative stability in price raises questions about refusal rates in the national survey. People who refuse to be interviewed may also be using drugs and do not wish to expose that use. Questions about use of illicit substances may prompt more unreliable answers regardless of confidentiality guarantees (Sidney 1990).

The surveys mentioned also attempt to measure coincident use of other drugs, legal and illegal. As the 1982 NAS report points out it is important to be cautious in interpreting information. Correlation between variables is not necessarily indicative of causative relationships. Alcohol is the single most prevalent drug used by adolescents, followed by tobacco and then marijuana. The combination of alcohol and marijuana may be more prevalent than surveys report (Norton 1988) with accompanying deleterious effects not previously estimated.

While it is likely that individuals using heroin have also used alcohol, marijuana and tobacco, there is no evidence to support a causal relationship. Research continues to focus on characteristics of the person who uses drugs to discover causes and, therefore, prevention or treatment strategies. As the 1982 National Science Foundation report observes, "[t]here is no evidence to support the belief that the use of one drug will inevitably lead to use of any other drug" (47). However, use of multiple substances is not uncommon.

Another area of concern is the reported increased potency of marijuana or cannabis extraction available today compared with that present in the early 1970s. Since many of the effects of marijuana have been linked to dosage amounts, a high potency marijuana may pose new dangers to users. The 1982 NAS report shows a range of marijuana potencies from about 1 to 3 percent. Sinsemilla varieties were high, even to 11 percent. Hashish and resins had the highest potencies (NAS 1982,16). Marijuana available in the late 1980s is reported to be considerably stronger and may range from 5 to 15

percent, depending on the source (Mikuriya 1988). However, the same article points out that the range of potencies may not have increased from earlier times, that the same strength of product has been available. 1973 and 1974 test laboratory results show a wide range of potencies.

## EFFECTS OF MARIJUANA

### Effects on the lungs and respiratory system

The several studies completed prior to 1982 contained conflicting results. Marijuana smoking in the United States is relatively recent for most of the population, so long-term effects have not been observed. In countries where marijuana or hashish smoking is generations old, it is usually also associated with tobacco smoking, so that effects of marijuana alone could not be isolated from the effects of tobacco.

Recent studies have shown, however, that marijuana smoking has a significant deleterious effect on the lungs and respiratory system. While acute periodic smoking results in bronchodilation, chronic smokers experience airway interference, an apparently reversible condition. Chronic heavy hashish use has been associated with respiratory ailments including bronchitis, sinusitis, asthma, and rhinopharyngitis (NAS 1982).

Marijuana smoke contains tars and many of the carcinogenic substances found in tobacco smoke, raising the risk of cancer. Marijuana is also smoked differently than tobacco. Puffs are held in the lungs longer and inhaled more deeply which results in respiratory effects from fewer marijuana than tobacco cigarettes. Marijuana cigarettes are also smoked without filters and occasionally contain contaminants. Among them are the salmonella bacteria and the aspergillus fungus, which can cause lung disease (Tashkin 1990a). There have also been instances of deliberate additions of PCP (phencyclidine), LSD or heroin to marijuana cigarettes.

Marijuana has been found to have an additive effect when used with tobacco. While each is harmful to the lungs, smoking both is much worse (Tashkin 1991a). Nonspecific airways hyperactivity was associated with smoking of both substances (Tashkin 1988). Another researcher examined lung tissue obtained from human subjects using an electron microscope and found evidence of epithelial cell damage in those who smoked marijuana or tobacco, with the most severe damage in those who smoked both (Fligiel 1988). Research also suggests that cellular changes occur in the absence of noticeable symptoms so that lung damage escapes clinical detection among the often young, otherwise healthy smokers (Tashkin 1990b).

Marijuana and tobacco smoke have been found to affect different areas of the lungs. The former has more effect on the central airway function and the latter on the

peripheral. Whether this is due to particle size or smoking behavior was not determined (Tashkin 1990b).

Another area of research concerns the defense system in the lungs. Several studies have shown that alveolar macrophages, which are part of the natural reaction to injury in the lungs, are present in increased numbers in those who smoke marijuana. Present also in those who smoked tobacco, the highest numbers and changes were noted in those who smoked both, reinforcing the observation that the two are additive (Tashkin 1990a, Barber 1988). Reporting on the results of several studies in humans, Tashkin notes that "... these results imply that marijuana smoking, like tobacco smoking, impairs the lungs' defense against microbial invasion, thereby increasing the tendency to respiratory tract infection" (1990a, 527).

### Effects on the cardiovascular system

Use of marijuana or THC has been shown to increase the heart rate and to increase blood pressure in some cases and decrease it in others. These effects in humans mimic stress and are neither enduring nor threatening to individuals with normal hearts and circulatory systems. The exact effects are somewhat dose dependent and also vary with method of administration and body position. Experiments with animals have sometimes yielded opposite results.

While not apparently threatening to healthy individuals, the increase stress on the heart could be dangerous to those suffering from coronary heart disease, hypertension, or heart failure. THC has been known to increase the work of the heart in several ways, none of which is advised for people with unhealthy hearts.

Long-term effects of marijuana on the incidence of coronary heart disease in chronic users have not been determined. The population of individuals who used marijuana during the 1960s and 1970s is just now entering the age at which these effects might appear. THC has been linked to changes in the EKG of both healthy individuals and those with coronary heart disease.

### Effects on the immune system

Experimentation with animals has suggested that a "mild, transient, immunosuppressant effect" is associated with delta-9-THC (NAS 1982). However, the data is inconclusive, and attempts to replicate many of the experiments have been unsuccessful. Experiments with animals and animal tissue have been criticized because of the very high doses of THC administered (Hollister 1988). Experiments with humans have been contradictory. There is no evidence to show that marijuana use increases the likelihood of infection in humans.

However, caution has been recommended because even mild effects on the immune system could be a



problem for individuals suffering from immune-related diseases, such as AIDS. Likewise, patients undergoing chemotherapy for cancer have lowered immune response.

## Effects on the reproductive system

This extremely complex area of investigation has also yielded conflicting results. Some effects demonstrated in animals have been questioned because of the large dose administered and the method of application, which as of 1982, was generally injected intraperitoneally. However, there is evidence that marijuana has some effects on the human reproductive system.

In human males, marijuana can reduce sperm count and sperm motility among chronic smokers, although no effect on fertility has been shown. These effects are modest and reversible. Plasma testosterone levels were decreased in several studies, along with follicle-stimulating hormone and luteinizing hormone, but other studies had conflicting results. Additional research is required in this area (NAS 1982). It was also found, in studies on primates, that the effects were reversible and mitigated by tolerance developed by users over time.

Most of the research on the effect of marijuana or THC on the female reproductive system has been on animals, of which the monkey may give the most reliable results when making comparisons to humans. In monkeys, THC administration was found to affect ovulation and the menstrual cycle. THC can cross the human placenta and, therefore, potentially affect the developing embryo. However, a protecting mechanism is suggested by lower levels in the plasma of the umbilical cord than maternal plasma at birth (US HSS 1987). High doses of THC administered in animals has demonstrated damage to the developing embryo, increased birth defects, and increased loss of offspring at or shortly after birth. Evidence of birth defects in humans attributable to marijuana smoking is lacking.

Research in this area is complicated by the use of several substances during pregnancy. When marijuana is used, it is often combined with alcohol, tobacco, and other drugs which makes it difficult to isolate the effects of any one substance. As discussed earlier, the additive potential exists here as well. Studies conducted in the early 1980s of mother/child pairs found that marijuana use was associated with lower birth weight and length.

The effects of marijuana on cells in the lungs were discussed in an earlier section. Smoke and tar have been shown to have a mutagenic effect in tissue cultures, both human and animals. THC alone has not been linked to mutagenic effects. THC has also not been linked to broken chromosome chains in humans, but there is evidence that it can affect the chromosome segregation during cell division, although this is inconclusive.

## Effects on the brain

Recent research, which identified the receptors and locations in the brain, has solved several important pieces of the puzzle and will likely expand or clarify the findings discussed below. These are based on the National Academy of Sciences 1982 report and augmented by the 1987 Second Triennial Report to Congress.

There have been no reliable research findings to support changes in the gross morphology of the brain. Computed tomography scans have given an indication of such changes. Electron microscope scanning of monkey brains in one experiment revealed changes, often mentioned in popular literature on marijuana. These findings ("... alteration in synaptic cleft width, increased density of synaptic cleft material, a decrease in volume of rough endoplasmic reticulum" [NAS 1982, 81]) are questionable because of flaws in the methodology, the small number of animals, and lack of replication. However, the possibility of these kinds of changes exists and warrants additional investigation.

Changes in waking brain wave patterns as a result of short-term use of marijuana have been observed. Most often alpha waves are increased, although some reduction has also been noted. Tolerance occurs after continued exposure to marijuana, mitigating these effects. These effects are apparently reversible with no long-term change noted. EEG readings taken during sleep also indicate changes, although not perceived in small dose administrations. The effects included shorter time in REM sleep and fewer eye movements. After marijuana was removed, REM sleep increased above the norm and more eye movements were observed. The experimenters also reported withdrawal symptoms including irritability, increased reflexes and mild agitation.

Two studies using electrode implants in the deep brain areas of monkeys showed changes which persisted for several months after marijuana was withdrawn. Although there are questions about the reliability of these studies, the few subjects, and species differences, this suggestion of possible persistent effects should be investigated.

Finally in this area, changes have been noted in actions of the chemical neurotransmitter acetylcholine. Small doses of THC reduce the actions of acetylcholine in the hippocampus. This only occurs with cannabinoids, among the drugs tested, and may be related to memory deficits (discussed below).

## Effects on learning and memory

Marijuana use has long been associated with impairment of short-term memory, especially noted on tasks requiring attention. Results of experiments involving memorization and recall of a series of numbers, for



example, correlate marijuana use with poorer performance. There is evidence as well that this is state-dependent, that is information learned under the influence of marijuana is best recalled while under the influence. The hippocampus is associated with learning and memory and, as mentioned earlier, marijuana receptor sites are concentrated in this area. Working with animals, one recent research project found that processing of sensory information, consistent with hippocampus function, was impaired under the influence of THC. Results suggested this was dose-dependent and was worse in tasks requiring the most attention (Deadwyler 1990).

Marijuana also affects the quality of oral communication, probably as a result of its impairment of short-term memory. Another reported effect is a distorted sense of time, so that subjects report an impression of a greater time passage than has actually occurred.

Research on the effects of chronic marijuana use on learning is limited. It is difficult to isolate marijuana use from other behaviors or circumstances, such as other drug use. A recent report, however, summarizes preliminary results of a study which attempts to assess the effect of heavy marijuana use over time (Block 1990). The adult subjects were matched on a number of variables including performance on their 4th grade Iowa Tests which were retrieved for this comparison. Performance on tests measuring verbal expression and mathematical skills were lower among chronic marijuana smokers than non-smokers. Test scores of those who used marijuana moderately were not significantly lower. None of the subjects used marijuana during the experiment. While not conclusive, this does suggest a possible effect warranting further research.

## Effect on perceptual and psychomotor functions

As with learning and memory, the acute effects (after a single dose) of marijuana or THC has been shown to impair perceptual and psychomotor functions. The effects of chronic use have been inadequately studied. Continuing long-term impairment have not been demonstrated, although it has been shown to persist several hours after intoxicating effects have stopped.

Marijuana and THC also impair coordination, tracking, and sensory and perceptual skills. The effects have been dose-dependent. Some of this impairment may be the result of decreased short-term memory functioning.

Simulator tests and performance on a test course have shown impaired driving performance. Two studies also reported impaired performance after the intoxicating effects of marijuana had ceased — one measuring driving performance several hours after smoking and another measuring performance of pilots in a simulator a full 24 hours after smoking (US Dept HHS 1987).

Marijuana is considered to be a risk factor in driving accidents. However, statistical evidence of marijuana use alone as cause of increased number of accidents is ambiguous. The strongest correlation is between drinking and driving. Marijuana and alcohol when used together have been shown to have an addictive effect, and blood tests from automobile accident fatalities have shown alcohol alone and alcohol in combination with marijuana to be prevalent. It has also been estimated that 90 percent of marijuana users also use alcohol (Gieringer 1988).

## Psychological effects

Marijuana is usually used because of its intoxicating effect, or "high." This effect varies with a number of factors including the experience of the user, the setting, dosage, and method of administration. The effect may be stressful, as has been reported by some users. Effects on interpersonal behavior are inconclusive. Marijuana is not thought to contribute to aggressive behavior, rather having a sedative effect.

Several negative reactions have been reported. These range from mild discomfort to more severe syndromes. However, these conditions could be dependent on the psychology of the user. Among the anxieties or panic reactions experienced are acute panic, paranoia, hallucinations, confusion, and fearfulness. Some report even more intense disorientation, even acute brain syndrome, or delirium. This condition was reported only after prolonged, heavy, regular use. The symptoms were found to disappear after drug use was discontinued.

Marijuana use has also been associated with "flashbacks" of hallucinogenic experiences from previous LSD use. Some patients who have suffered from mental illness report that marijuana interferes with their feelings of well-being. It has also been suggested that marijuana may interfere with some antipsychotic medications (US Dept HHS 1987).

There is not sufficient evidence in North America to substantiate the occurrence of cannabis psychosis, although it has been reported in Africa and Asia. Another common chronic effect attributed to marijuana use is "amotivational syndrome" which includes apathy, loss of ambition and energy, and inability to concentrate. It is not known how much of this is attributable to marijuana. "Many troubled individuals seek an escape into use of drugs; thus, frequent use of marijuana may become one more in a series of counterproductive behaviors for these unhappy people" (NAS 1982, 125).

## TOLERANCE AND WITHDRAWAL

Tolerance has been shown both in human and animal subjects. One researcher reports having found neurons in the central nervous system which became sensitized to

THC after several hours of exposure. This was dose-dependent and site specific and there was no cytopathology upon cessation (Martin 1988).

There is also evidence that tolerance is subject to learning. Tolerance in animals developed according to the experimental condition — quickly when the condition or test parameter was negative, and slowly when it was positive. Also, tolerance does not develop in all cases (Compton 1990).

Withdrawal has been shown, but is generally associated with heavy, chronic use. Even then symptoms are mild and include irritability, agitation, insomnia, and EEG changes. These symptoms peak at 30 hours after deprivation and disappear at 90 hours (NAS 1982). In the laboratory, withdrawal in animals has also not been strongly demonstrated. It is difficult to measure withdrawal. One method of detection is to allow self-medication during the withdrawal period. Laboratory animals have not been shown to respond strongly to this opportunity following deprivation (Compton 1990). The evidence for physical dependence on marijuana is weak.

## THERAPEUTIC APPLICATIONS AND POTENTIAL

Several potential therapeutic applications of marijuana, or more specifically the various cannabinoids, have been suggested. Among the applications are treatment of glaucoma, use as an antiemetic to control nausea and vomiting associated with chemotherapy, as an appetite stimulant, anticonvulsant, and for control of spasticity.

Synthetic THC has been provided for some of these conditions as well. The side effects, however, of increased heart rate, lowered blood pressure, and psychotropic effects preclude use of marijuana and THC in some cases. Many patients cannot tolerate smoking. Other unpleasant effects reported are sedation, dry-mouth, red eyes, dizziness, and the feelings of anxiety mentioned earlier (Talan 1990).

## MARIJUANA ABUSE AND TREATMENT

The discussion so far in this paper has focused on the known effects of marijuana. Throughout, the psychological state of the user has been mentioned mostly as a matter complicating conclusions of pure scientific research. However, marijuana is an intoxicant. It alters perceptions of time, interferes with short-term memory and learning and is associated with altered psychological states. It is not necessary to prove physical dependence to treat psychological dependence as a serious condition with viable treatment options.

Increase in the use of urinalysis to detect drugs in the work place has prompted many individuals to come forward for treatment of abuse, according to one re-

searcher (Roffman 1988). In the first two weeks of a research effort, over 200 individuals in the Seattle area responded to an advertisement for assistance for treatment for dependency on marijuana. This suggests that there are people, some of whom have used marijuana without perceived deleterious effects for years, who are having difficulty terminating its use.

Researchers continue to try to identify the conditions which lead to chronic use and abuse of marijuana, especially among adolescents, where the behavior might be perceived as benign. Some of the correlates (not causes) of daily use discussed by the Committee of the Institute of Medicine are residence in urban rather than small communities, gender (males used more frequently), poor school performance, absence of religious commitment, use of other drugs, single status, and living away from parents (NAS 1982, 41).

Clearly many factors could be concurrent with both marijuana use and the correlates mentioned above. Gabany and Plummer (1990) identified five factors which were associated with student perceptions of beginning marijuana use: maturational difficulties, excessive pressure, parental failings, rebelliousness, and societal/institutional weaknesses. The intent is to isolate conditions which can benefit from improved educational efforts. These and similar explorations of correlates of beginning and continued use of marijuana and other drugs can improve prevention and treatment strategies. (See also Capuzzi 1982, Mayer 1989, Rob 1990, and Dembo 1991.)

Treatment strategies to deal with many of these complex factors as well as possible concurrent abuse of other substances are numerous. Group, family, and individual counseling is suggested by some. Prevention education is emphasized, which includes the societal message that drug use is harmful. The 12-step treatment approach similar to that used in Alcoholics Anonymous has also met with success (Miller, 1989).

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# For the Record . . .

## Health Care Reform: Taking Focus

Senator Frank H. Murkowski

The challenge of quality health faces Alaskans and all Americans concerned about the direction our state and national reform efforts will take. It is a challenge not only to ensure that federal and state reform efforts do not work at cross purposes, but to make sure that reform leads to improvement — that we improve on the highest quality health care system in the world, rather than damage it with overregulation and untested reforms.

Many look toward the government at both the state and federal levels and ask their legislators to solve problems of cost and access. States and the federal government can do a great deal through Medicare and Medicaid reforms, changes to the tax code, antitrust and malpractice reforms and improving coordination among the federally run health delivery systems like the Department of Veterans Affairs, the Indian Health Service and the Department of Defense.

Alaska is extremely dependent on federal sources to pay for health care services. It is important that any reform plan not sever the crucial federal ties that keep these programs running, and that we keep an open mind about further integration and coordination of federal health care programs.

But the focus of reform also must include the consumer — the individual — where the true responsibility for health care lies. Congress' goal of assuring universal access and controlling costs should be grounded on the premise that individuals are ultimately responsible for choosing and paying for their health care. Our mandate should be to provide incentives for individuals to become better consumers to encourage people to make cost-conscious health care choices consistent with personal needs.

Senator Don Nickles, R-Okla., has introduced a bill, which I have cosponsored, that stresses choice and individual responsibility. The "Consumer Choice and Security Act," would use personal tax credits to help individuals and families pay for their health benefits.

Insurance coverage would be guaranteed, no one would be denied coverage based on preexisting conditions, and insurance would be tied to the individual, not the employer. At a minimum, each person would be required to have catastrophic health insurance. Beyond this, an individual would be able to purchase additional coverage from competing plans and would receive a tax credit.

The Nickles plan favors limited federal government

involvement in health care. It builds on the strength of our current system: plentiful doctors and facilities, patient's freedom of choice in selecting physicians, and accessible high-tech, quality health care.

What does this mean for Alaska? It means consumer choice and responsibility; health care made affordable by increased competition in the insurance market; tax credits provided in each paycheck; and choice of benefits based on individual needs, which, as we know, are different for a single 21-year-old than for a family of four. Furthermore, it means states would be allowed to choose their own course, while making a limited number of required federal reforms facilitating universal, affordable coverage.

In contrast, the Clinton proposal, and other proposals

that combine big government with price controls, are based on the faulty premise that sole-source health care funding — whether at the state or national level, is consistent with using competitive market principles to control costs and preserve choice. The Clinton plan requires the National Health

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. . . if you think health care costs are high now, wait until you see what they cost when they are "free."

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Board to establish a national health budget and gives health alliances authority to cap premium prices and regulate services. Heavy top-down control combined with government subsidies for low-income workers, means that the benefits of market competition would be lost. The end result would be an increased demand for services and a diminishing number of providers (which Alaska can ill afford) — possibly leading to a system of rationed care.

In addition to being overly optimistic about cost savings from its reforms, the Clinton plan's ideological bias run counter to what Alaskans expect from government. Alaskans do not want a new bureaucratic monster (The Clinton plan established no less than 77 new governmental entities) controlling costs and access. We do not believe in a "one-size-fits-all" philosophy. And we do not want to adhere to a requirement that states alter themselves in order to fit into a federally mandated puzzle that does not suit our needs. These beliefs are supported by information I have gathered from Alaskans that indicate that 75 percent are unwilling to pay

(continued on page 126)

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# Telemedicine Technology

Senator Ted Stevens

In West Virginia they call it "Mountaineer Doctor Television."

In North Carolina, a university hospital supplies medical care via television in the Department of Corrections in another city.

Our own U.S. military provides quality medical care to patients in remote places throughout the world through modern electronic technology.

There's a word for this process: telemedicine. No doubt, some of the readers of this publication have already utilized telemedicine technology.

While it's in relatively early stages in the civilian medical community, telemedicine isn't new to military health providers. Over the past decade, military doctors, working with Georgetown University Medical Center, have made telemedicine a standard of care for military personnel stationed in distant posts. Their success proves that the quality of health care in our state, particularly in rural Alaska, could be significantly expanded through the use of telemedicine.

Let's look at some of the Department of Defense programs:

Currently, the Department of Defense is using telemedicine to support our troops in Croatia and Macedonia. U.S. military doctors stationed in the former Yugoslavia are consulting on a regular basis with specialists at Walter Reed and other large military hospitals.

The results are impressive: a medical officer in Macedonia estimates that teleradiology and other imaging technology has enabled him to reduce medevacs by more than half. He said access to the technology helps him to work with greater confidence, and the troops have more faith in his diagnoses.

We have an example of the military's use of telemedicine closer to home, in Adak. The Naval base on the Aleutian chain will no longer have a full-time physician because of Department of Defense (DoD) force reductions. The base will rely on telemedicine for medical support.

And a little further down the road, the Army's Madigan Medical Facility near Tacoma, Washington, and McDonnell Air Force Base in Kansas are linked to the DoD's teleradiology network.

In addition, the Veterans' Administration is implementing a digital archiving system for medical records and images. Hospitals will be able to access these records instantly and refer difficult cases to specialists across the country.

The director of Georgetown University's division of Imaging Science and Information Systems, Dr. Seong K. Mun, visited remote areas of our state, including the

Yukon-Kuskokwim region and Southeast, to explore the use of telemedicine to support physicians and community health aides.

In a preliminary report on his Alaska visit, the Georgetown doctor said, "The needs are great and the challenges are significant, but the technology exists for dramatically improving the current situation, particularly at the village clinic and regional hospital levels."

The preliminary report lists possible improvements that telemedicine could provide for rural health care delivery. Among the suggestions:

- Computerization of the patient information form, to reduce errors and time-consuming redundant data entries. This would develop an electronic patient record for use throughout our state.

- Installation of a digital network capable of transmitting patient data, E-mail and still pictures from village clinics to regional hospitals. This would also give Native Alaskans and rural residents access to national information resources such as Internet.

- Enhancement of the information base at regional hospitals, which would improve the exchange of patient data between Community Health Aides and hospital-based physicians.

- Establishment of teleradiology service between regional hospitals and an experienced major medical center.

- Development of an electronic version of the "community Health Aides/Practitioner Manual," and improved access to distance learning programs for Community Health Aides.

When decisions must be made quickly, as in the case of emergency and trauma care, telemedicine can minimize delays. Patient data can be transmitted immediately to larger medical centers with access to specialists and additional medical technology.

Wide-scale telemedicine in our state will require accessibility to affordable satellite time. Alaska's geography makes it almost impossible to utilize fiber-optic cable in many parts of our state.

Alaska is fortunate not to be subject to Inter-Local Access and Transport Area (Inter-LATA) tariffs, which could hinder the development of telemedicine. But many other states are subject to the tariffs, which are obstacles to extending telemedicine networks. Telecommunications reform may be the answer to removing the barriers the tariff puts up.

Congress's involvement may be required in another area, as well. It's likely that the formulas for Medicare and Medicaid will have to be updated to include

(continued on next page)



# California Large Loss Trend Study

California's tort-reform statutes have kept malpractice awards in check, but haven't reduced the number of lawsuits filed. So reports Medical Underwriters of California in its 1993 update of the California Large Loss Trend Study, an annual survey of \$100,000 + California medical malpractice verdicts and settlements. The study describes the benefits of California's Medical Injury Compensation Reform Act (MICRA): present and average value indemnity were lower than in previous years, and MICRA's limits on noneconomic damages and its requirements for collateral source offsets and periodic payments reduced total costs.

## Other findings:

- Sixteen birth-injury cases were reported in 1993, down from 21 in 1992, but still almost double the average over the past five years. Obstetrician-gynecologists continued to lead the field of chief defendants, a ranking they've had since 1989, with involvement in 26 cases. The next highest specialty sued was general surgery, followed by orthopedics.

- Most cases (53%) were settled in lump-sum awards, and 42% went to jury verdicts. Only 4.5% were structured settlements.
- Failure to diagnose or treat continued to be the leading allegation, at forty-one percent of cases.
- The number of lawsuits in non-hospital settings jumped to 43 in 1993, from an average of 30 in the preceding five years. More than 60% of the cases and total indemnity resulted from injuries sustained in settings other than the hospital operating room, emergency room, or labor and delivery areas.

For more information about the report, contact Ron Neupauer, Medical Underwriters of California, 6250 Claremont Avenue, Oakland 94618. Telephone: 800/227-4527. FAX 510/654-4634. Copies are \$5 each.

*Medical Underwriters of California manages Medical Insurance Exchange of California, one of the first doctor-owned professional liability insurance carriers formed in the malpractice crisis of the mid-seventies.*

(continued from previous page - Telemedicine Technology)

reimbursement for remote consulting. As a matter of fact, the Health Care Financing Administration has tentatively approved a demonstration project on reimbursement for remote consulting.

NASA, the National Aeronautics and Space Administration, is a pioneer in fostering the development of telemedicine. In the early 1970s NASA used space technology to bring telemedicine to a remote Arizona Indian reservation. NASA experts estimate that \$36 billion a year in health care costs could be saved by the application of nationwide telemedicine technology.

The Office of Technology Assessment (OTA) plans a two-year study to identify the steps necessary to ensure that American Indian reservations and Alaska Native communities will benefit from advances in telecommunications technology.

There is movement on many fronts to make telemedicine available to more Americans, with federal grant and other assistance programs for telemedicine already in place.

Individual agencies, such as the Rural Electrification Administration of the Department of Agriculture, the Department of Energy's Office of Scientific Computing, the National Telecommunications and Information Administration of the Department of Commerce, NASA, the VA and the Department of Defense are among those with ongoing programs.

Other agencies using telecommunications technology for health care programs include the Department of Health

and Human Services, the Centers for Disease Control and Prevention, the Food and Drug Administration, the National Institutes of Health, the Office of Rural Health Policy and the Health Care Financing Administration.

In Alaska, our remote clinics and community health aides suffer from many of the same problems that the military does in the field. They are isolated, often with no access to specialists or sophisticated diagnostic tools.

Just as our state's earliest communications systems grew from the military's experience and pioneering, so can telemedicine benefit from the military's use of it today.

And, as we work toward a nationwide system, congress must be careful not to dictate hardware and software solutions that could lock telemedicine into outdated technology or suppress innovation.

There is much Congress can do to encourage the careful development of telemedicine. It will be important for emerging networks to be able to rely on open systems and not be restricted from using products and services from a competitive marketplace.

I have joined in sponsoring S.1770, the Health Equity and Access Reform Act (HEART), which includes telemedicine-related provisions. It calls for the standardization of patient records, uniform billing procedures and guidelines for privacy and confidentiality. Another bill I support, the Consumer Choice Health Security Act, offers grants to build links between rural and urban medical facilities.

# Sexually Speaking . . .

## Sexual Desire Disorder as a Primary Diagnosis

Mary Cavalier, M.S.<sup>(1)</sup>

The referrals I often get from physicians are: women with dyspareunia or vaginismus, and men with premature ejaculation or impotence. The typical treatment protocol for these diagnoses can be addressed in short term sex therapy. Recently, though, I have had to challenge my paradigms associated with the presenting "primary" diagnosis. This challenge came about by several cases which, despite the high motivation of the client, failed with the traditional treatment. Allow me to present a case study which illustrates this phenomenon (note: the demographics of the case has been changed to protect the confidentiality of the patient).

### CASE STUDY

A woman in her mid-forties presented to her doctor a case which appeared to be situational vaginismus. The patient had no problems having a pelvic exam. The patient did state that over a period of time it has become impossible to have intercourse with her partner. The doctor recommended the appropriate treatment protocol of using dilators in progression until the ability to have intercourse returned. During the 6-week follow-up session, the patient stated that "things have only gotten worse." The doctor referred her to me.

When I did a full sexual history on the patient a key element stuck out. The patient reported that intercourse had been painful for a number of years (with no apparent physical source). She reported that she "white knuckled her way through intercourse." During this same period of time, a child was born and job demands increased.

I suggested a series of sensate exercises designed to slowly reintroduce the patient to the pleasure of the body. The patient would report never having time to do the exercises.

I began to realize that I was making an assumption. The assumption was that since it was a concern of the patient there must be desire. Wrong! What had happened over time with the combination of painful intercourse and increased stress, was that the patient lost all interest in sex. The primary issue was diminished sexual desire. This explained why the traditional treatment protocols failed. There was no desire to motivate change!

### TREATMENT

Treatment of sexual desire disorders is often challenging and long term. There is some debate in the sexology field of whether in-depth psychotherapy is necessary. For myself, I rule out sexual abuse first. If sexual abuse had occurred, those issues must be addressed first. If sexual abuse is not an issue, then I approach treatment via a cognitive restructuring model. By working with the individual and couple, we examine the beliefs associated with being sexual. For example in the case presented, the belief was "sex is always painful." Then there is a systematic evaluation of looking at the facts that lead to the belief(s). During this process, specific behaviors which sabotage recovery usually manifests itself.

The patient begins to realize that diminished sexual desire is a learned behavior and what has been learned can be changed. Along with the cognitive restructuring is the introduction of the sensate exercises. Participation of the partner is key throughout the whole process.

### CONCLUSION

When a patient doesn't respond to the traditional treatment protocols of sex therapy, it is beneficial to rule out a primary diagnosis of sexual desire disorder. For if there is no desire, there is no true desire to change.

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<sup>(1)</sup> Robert Alberts, M.D. & Associates, 3340 Providence Drive, Anchorage, Alaska 99508

# History of Medicine in Alaska

A.B. Colyar, M.D.

Ardell B. Colyar, called A.B., was born in Oklahoma where he spent his childhood in several different towns, graduating from Eldorado High School. He attended Cameron Junior College in Lawton entering the Civilian Conservation Corps (CCC), one of the programs instituted by President Franklin D. Roosevelt to help ameliorate the Great Depression of the 1930s.

A. B. claims to have worked in the "chain gang", which built a 10-foot steel chain fence around a wildlife refuge in the Wichita Mountains of western Oklahoma. He stayed in the CCC, supervised by the Army, for four years, attending evening college and finally summer school to acquire credits to enter the University of Oklahoma Medical School.

Benita Morgan and A.B. Colyar were married at the end of his second year of medical school. They went to Des Moines, Iowa for a rotating internship at the Broadlawns Hospital. He remained for a year and a half of a surgical residency before being drafted. During this residency, he was involved in an experimental program for the treatment of syphilis.

He chose to enter the United States Public Health Service as a commissioned officer in venereal disease control, working with state health departments. Dr. Colyar was first at Augusta, Georgia and then at the Oklahoma Treatment Center in Rush Springs, which boasted of being the Watermelon Capitol of the World.

A treatment for syphilis was developed using Mapharsen, an arsenical compound. The drug was administered intravenously every eight hours for five to seven days. Since the old treatment took years to cure, this was an improvement. Later, penicillin was given intramuscularly several times a day for ten days.

At this time, it was possible to quarantine patients with syphilis and gonorrhea. Society had the right to treat; the individual did not have the right to refuse. Buses were sent to pick up known VD patients. At the end of the war, soldiers brought to discharge centers were not separated from service until their tests were negative for VD. Often it took two or three rounds of testing and treatment to establish a clean bill of health.

A. B. Colyar next became VD control officer for the Oklahoma State Department of Health. When the center became part of the university, he found himself Adjunct Professor of Venereal Disease Treatment and Education.

In 1948, he entered private practice for a brief period of time in eastern Oklahoma before rejoining the USPHS to gain formal training in public health. His time in the

Oklahoma health department was credited as one year. He finished at the Johns Hopkins University School of Hygiene and Public Health in 1950, earning a MPH.

Joining the Boston Regional Office which served all the New England states, he served as a consultant in chronic diseases to the Massachusetts Health Department as a mediator between the federal and state governments for grants, program funding, and technical assistance for tuberculosis, heart disease, cancer and diabetes.

When the regional office in Boston closed, he was assigned to New York City. After one year, he was offered a position in Alaska. He took it.

In Alaska, tuberculosis was rampant among the Natives. The Surgeon General, Dr. Thomas Parran, was asked to study the problem. The Alaska Native Medical Center had been built as a tuberculosis hospital with 53 beds for patients with tuberculosis. A plan to find cases had been developed but not implemented.

In 1948 an all-out effort was made. At the height of the program the Anchorage hospital, Harborview and Firland in Seattle, and the Tacoma U.S. Public Health Service Hospital were all pressed into service. In addition, village programs were devised, using two oral medications. Public health nurses trained chemotherapy aides to dispense the medicine, isonicotinic acid hydrazide (INH) and para-amino-salicylic acid (PAS). Medicine was stocked in 22 villages. Every family had its own supply. Unfortunately, a few children died when they accidentally ingested medicines.

The PHS mounted a survey by means of portable x-ray equipment. One doctor, regularly flown in by bush pilot, asked to lease an airplane since he could fly. The plane was found unused at Merrill Field. The doctor had found it too lonely flying by himself and had gone out on a commercial flight.

In those early days there was good cooperation among the various agencies. In 1955, the Bureau of Indian Affairs handed over medical services to the PHS, even allocating part of its budget.

It was the custom when visiting villages to stay with the school teachers. Dr. Colyar remembers being offered ice cream by one of them. He was hesitant since he had not acquired a taste for Eskimo ice cream (blubber and blueberries). However, it turned out to be regular vanilla ice cream, but it was frozen like a brick in the below zero temperatures.

To take every precaution, skin tests also were used. From these tests it was determined that approximately 25



percent of three-year-olds were infected. By 1964, the rate dropped to one or two percent. This was dramatic proof that tuberculosis could be treated. By 1965, the last designated tuberculosis bed was closed and all patients, except for the very enfeebled, were treated as outpatients.

A.B. Colyar became Director of the Arctic Health Research Center in Anchorage. Under his skillful guidance, investigations were conducted in epidemiology (under Bob Phillips and Jim Maynard), zoonotic and metabolic diseases, cold adaptation, sanitation and water quality. The cause of the diarrheal disease known as beaver fever (giardiasis) was discovered. Alaska mosquitoes were studied to see if they transmit disease. They do not. At Fort Yukon, state Fish and Game agents painstakingly examined 1,000 molting ducks to see if they carried viral disease. None was found. Dr. Milo Fritz was dispatched to the field to take out tonsils and treat otitis media. Natives were tested to find cases of sub-clinical mumps. There was work on tularemia and botulism.

Bob Raush, PhD, who had a wolf named Wolf, worked on St. Lawrence Island with *Echinococcus multilocularis*, an organism causing cysts in the lungs and liver. He developed a blood test for this disorder.

Edward Scott, M.D., and colleagues studied methemoglobinemia, a disorder in which blood cells do not carry oxygen efficiently. The Center cooperated with Greek scientists also working on this disease.

Laurence Irving wondered to what degree mammals could adapt to cold. He concluded that pigs could not.

Despite having twenty acres on Goose Lake the Center was moved to the campus of the University of Alaska in Fairbanks in 1967 because Senator Greuning felt an arctic research center should be in the Arctic. Just prior to this move, Dr. Colyar left the Center.

He went to the Industrial College of the Armed Forces at Fort McNair to study the economics of industry in relation to the national defense; then back to Oklahoma, where he served for four years as state commissioner of health.

Upon returning to Anchorage, he became the director of health for the Borough of Anchorage. When the city and borough were unified, he was director of the Anchorage Health Department.

From 1973 to 1977, he served on the National Advisory Board for Nurses' Training.

Finally, Dr. Colyar was in private practice for eight years with Drs. Morgan, Olson and Agnew before retiring in 1984. Since retirement, he has been involved with church activities, serving as president of the Alaska Baptist Convention.

A.B. and Benita Colyar have four daughters. She has been active with Girl Scouts, has served on the board of the Anchorage Concert Association and performed as a member of the Anchorage Community Chorus.

People who experienced the Great Depression seem to have developed a resiliency which enables them to adapt to changing situations. Such was the case with the able and engaging Dr. A. B. Colyar.

Gwynneth Gminder Wilson  
Alaska Medical Alliance

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# GLIMPSES OF ALASKAN MEDICAL HISTORY

Edited by Robert Fortuine, M.D.

## A look at hospital care in New Archangel (1835-1840)

A previous article in this series briefly described the medical care available to the workers of the Russian-American Company beginning in 1820. The initial eight hospital beds had to be increased during the first decade to more than twenty, to accommodate the expansion of the commercial operations at the capital. Baron von Wrangell, one of the Russian governors, noted in the early 1830s that the hospital was a "large, well-maintained building that was generously supplied with all the necessities."<sup>1</sup> Whenever possible the company employed two physicians at the hospital, but recruitment difficulties precluded such a luxury most of the time. Much of the routine medical work instead was carried out by *feldshers*, almost a type of physician's assistant, usually mixed Russian-Alutcs who had been trained at the hospital.

Many of the doctors and *feldshers* who served in the early years have left little trace, a notable exception being Dr. Blaschke, introduced in the previous article. Within a month of his arrival, as mentioned, the devastating smallpox epidemic had absorbed all his energies. The widespread sickness in the capital made it necessary for him not only to make use of every available room at the hospital, but also to establish an additional small facility for women a short distance from the main building. Yet, a visitor—Lieutenant Belcher of H.M.S. *Sulphur*—remarked in 1837, perhaps near the peak of the epidemic, that the hospital was "comparatively clean" and comfortable.<sup>1</sup>

The following brief excerpt is from Blaschke's own description<sup>2</sup> of the hospital during the time he was stationed at the capital.

"The men's hospital building is about nine fathoms long and eight fathoms wide. A hallway extends from the entrance, which is concealed by the long dimension, to the right of which is a room designated for the reception of patients and two wards; on the left side of the hallway is the kitchen, the pharmacy and a third ward. This corridor extends beyond the house and is joined on the left by the bath house. At the end of it are located the latrines, which are washed by seawater at high tide. In the rather spacious upper floor the medicines and the wooden chests for storing soiled linens are kept, and there is a place for hanging and drying the herbs collected in the region. Next to the kitchen is a small room for the surgeon. On either side of the entrance are small rooms—one where the clean linen is kept, the other for receiving the jars and food containers brought on some days from

the warehouse. The hallway can be heated and the wards are rather spacious and agreeable, and lighted with by rather large windows. The floor is painted an olive color. The twenty-four beds are painted olive-green, but if the number of patients is increased, four more beds are added, and six patients can be placed in the reception room. The mattresses and pillows are stuffed with coconut flax, there being insufficient horsehair available. The blankets are woollen and the sheets of linen; they are changed everywhere once a week. . . . The pharmacy is rather spacious and well arranged; in a room separated from the antechamber is a furnace for distillations maintained in a sand bath and for preparing chemical compounds. Potions, plasters, and other remedies are warmed in the kitchen. . . .

Aside from the physician, the following are engaged on hospital duty in delivering care and attention to the sick: a naval surgeon of upper grade who under the physician's supervision largely performs the duties of a pharmacist; a Creole assistant qualified at the same educational level as the surgeon, and five or six Creole students who are instructed with the necessary information by the surgeon; these have the night duty on the wards, and assist the pharmacist and patients. Others include one supervisor, a cook and two helpers, and a midwife with one assistant; . . .

"The daily number of patients differs markedly according to the season, weather, and so on. There are rarely less than twenty patients in the hospital, and less than ten ambulatory patients. The number of the former, however, often grows to thirty and the latter to forty or more. All patients, including women and children, under the most favorable conditions rarely reach a total of forty in the summer, but in the fall, the winter, and sometimes even the spring, the number exceeds eighty or a hundred."

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# From Out of the Past — Over 30 Years Ago

Gloria K. Park, M.D.

## Roster of Physicians in Private Practice in Alaska —1963

### ANCHORAGE-SPENARD

Population — 54,753

Hospital Beds: 125 (Providence Hospital — Catholic)  
40 (Presbyterian Community Hospital)

BARTKO, HAROLD - General Surgery  
2220 E. Northern Lights Blvd.

BEIRNE, MICHAEL F. - Pathology  
207 E. Northern Lights Blvd.

BILLINGS, ROBERT P. - General Practice  
209 Fireweed Lane

BLANKINSHIP, GILBERT - Internal Medicine  
207 E. Northern Lights Blvd.

CATES, VERNON A. - General Practice  
825 L Street

CAUGHRAN, WILLIAM R. - General Practice  
Box 4-848, Spenard Medical Clinic

CHAO, CHI-MEI HWANG - Anesthesiology  
1145 12th Ave.

CHENOWETH, CHARLIE E. - E.E.N.T.  
Mt. McKinley Bldg.

COIN, JAMES WALTER - Radiology  
Providence Hospital

CRAWFORD, GLENN B. - General Practice  
203 W. Fireweed Lane

DEAL, CLYDE F. - General Practice  
207 E. Northern Lights Blvd.

DRAKE, DUANE L. - Radiology  
207 E. Northern Lights Blvd.

FISH, WINTHROP - Internal Medicine  
501 L Street

FITZPATRICK, JAMES J. - Internal Medicine  
718 K Street

FRITZ, MILO H. - E.E.N.T.  
1027 Fourth Ave.

HALE, GEORGE E. - General Surgery  
501 L Street

HARRELL, R.E. - General Practice  
520 E. 14th Ave.

HEPPLER, LAWRENCE - Contract Physician  
c/o FAA, Box 440

HILLMAN, FREDERICK J. - General Surgery  
207 E. Northern Lights Blvd.

HOMAY, ALAN - General Practice  
207 E. Northern Lights Blvd.

IVY, WILLIAM H. - Obstetrics & Gynecology  
825 L Street

JACKSON, MARCELL - General Practice  
825 L Street

JOHNSON, CALVIN T. - General Practice  
1104 G Street

JONES, WARREN - General Practice  
207 E. Northern Lights Blvd.

KETTELKAMP, DONALD B. - Orthopedic  
Surgery, 742 K Street

KIESTER, THOMAS E. - Orthopedic Surgery  
207 E. Northern Lights Blvd.

KOENIGER, PETER J. - Obstetrics &  
Gynecology, 825 L Street

KRAFT, EDWIN C. - General Surgery  
718 K Street

LANGDON, J. RAY - Psychiatry  
207 E. Northern Lights Blvd.

LANGSTON, DON VAL - Pediatrics  
207 E. Northern Lights Blvd.

LEONG, RUDY H. - General Practice  
207 E. Northern Lights Blvd.

MADDOCK, WILLIAM O. - Internal Medicine  
825 L Street

MARGETTS, LESTER H. - General Surgery  
825 L Street

MARTIN, ASA L. - General Practice  
718 K Street

MATTHEWS, WENDELL C. - Contract Physician  
c/o FAA, Box 440

MEAD, PERRY A. - Neurosurgery  
825 L Street

MILLS, WILLIAM J. JR. - Orthopedic Surgery  
742 K Street

MONTMORENCY, FRANK A. - Urology  
207 E. Northern Lights Blvd.

MORGAN, ROYCE - General Practice  
207 E. Northern Lights Blvd.

OGDEN GLEN - Radiology  
Presbyterian Community Hospital

O'MALLEY, JAMES E. - General Practice  
529 I Street

ORMOND, LOUISE - Internal Medicine  
207 E. Northern Lights Blvd.

PENNINGTON, JOHN A. - Anesthesiology  
2101 Turnagain Parkway

PHILLIPS, FRANCIS L. - Chest Diseases  
2220 E. Northern Lights Blvd.

RABOURN, WILLIAM O. - General Practice  
Box 1146, Spenard

RADER, WILLIAM - Psychiatry  
305 Fifth Ave.

RENN, A. CLAIRE - Obstetrics & Gynecology  
825 L Street

ROMIG, HOWARD G. - Gynecology  
718 K Street

ST. JOHN, CHARLES F. - General Practice  
207 E. Northern Lights Blvd.

SEDWICK, JACK D. - General Surgery  
718 K Street



SHELTON, JOSEPH H. - Ophthalmology  
825 L Street  
SHOFF, MAILON J. - E.E.N.T.  
825 L Street  
SHOHL, ROSALIE - Anesthesiology  
207 E. Northern Lights Blvd.  
SHOHL, THEODORE - General Surgery  
207 E. Northern Lights Blvd.  
STARR, MERRITT P. - Internal Medicine  
825 L Street  
STRAUSS, FREDERICK - Pathology  
Providence Hospital  
SYDNAM, NANCY E. - General Practice  
140 E. Fifth Ave.  
TOWER, ELIZABETH - General Practice &  
Adolescence, 718 K Street  
TOWER, JOHN C. - Pediatrics  
825 L Street  
WALKOWSKI, A. S. - General Practice  
429 Fourth Ave.  
WHALEY, HELEN S. - Pediatrics  
825 L Street  
WHALEY, ROBERT D. - Internal Medicine  
825 L Street  
WICHMAN, GEORGE B. - Orthopedic Surgery  
718 K Street  
WILKINS, ROBERT B. - Internal Medicine  
718 K Street  
WILSON, RODMAN - Internal Medicine  
207 E. Northern Lights Blvd.  
WRIGHT, VIRGINIA - Psychiatry  
529 I Street  
ZARTMAN, HARVEY F. - Pediatrics  
825 L Street

## FAIRBANKS

Population — 13,311  
Hospitals Beds — 70 (St. Joseph's Hospital-Catholic)  
BUGH, C. WILLIAM - General Practice  
1255 Airport Way  
CLARK, GEORGE - Anesthesiology  
Box 167, College  
DEELY, NICHOLAS - Pediatrics  
Box 1330  
EVANS, RAYMOND C. - General Practice  
Box 1330  
FAULKNER, ROBERT F. - Ophthalmology  
Box 1330  
FENNER, JOHN LEWIS - General Practice  
Box 1330  
HAGGLAND, PAUL L. - Orthopedic Surgery  
Box 1330  
HEAVRIN, LAWRENCE - General Practice  
1007 Noble Street  
JOHNSON, JOSEPH K. - General Surgery  
Box 1330  
LEIH, GOERGE G. T. - General Practice  
1007 Noble Street  
LUNDQUIST, JAMES A. - General Practice  
1007 Noble Street  
MARROW, CHARLES T. - Internal Medicine  
Box 1330  
MEYER, EDWARD D. - Internal Medicine

Box 1330  
RIBAR, JOSEPH H. - General Practice  
Box 1330  
SCHIAIBLE, ARTHUR J. - General Surgery  
Box 1330  
STORRS, HENRY G. - General Surgery  
Box 993  
TATUM, DONALD E. - Internal Medicine  
1007 Noble Street  
WESTON, JOHN I. - General Practice  
411 Fourth Avenue

## JUNEAU-DOUGLAS

Population — 7,839  
Hospital Beds — 79 (St. Ann's Hospital-Catholic)  
AKIYAMA, HENRY I. - Internal Medicine  
188 S. Franklin  
DALTON, JOHN A. - General Practice  
188 S. Franklin  
GIBSON, JACK W. - Pediatrics  
188 S. Franklin  
MILLER, E. CUMMINGS - General Practice  
188 S. Franklin  
RAY, E. STANLEY - General Practice  
Box 2627  
RAY, HOMER F. JR. - Psychiatry  
303 Coleman Street  
REIDERER, JOSEPH - General Practice  
I.O.O.F. Bldg.  
RUDE, JOSEPH O. - General Practice  
I.O.O.F. Bldg.  
SMALLEY, ROBERT R. - General Surgery  
188 S. Franklin  
WHITEHEAD, WILLIAM M. - General Practice  
188 S. Franklin  
WILDE, HENRY - Internal Medicine  
188 S. Franklin

## KETCHIKAN

Population - 6,483  
Hospital Beds — 80 (Ketchikan General Hospital)  
CARR, RALPH W. - General Practice  
Box 359  
SALAZAR, LOUIS - General Practice  
Box 359  
SMITH, PHYLLIS E. - General Practice  
Box 86  
WILSON, A. N. - General Practice  
Box 1989  
WILSON, JAMES A. - General Surgery  
Box 1989  
WILSON, A. N. JR. - Internal Medicine  
Box 1989  
WINTON, ERVIN O. - General Practice  
2433 First Avenue

## SITKA

Population — 3,237  
Hospital Beds - 23 (Sitka Community Hospital)  
MOORE, PHILIP H. - Orthopedic Surgery  
Box 810  
MOORE, TILLMAN JR. - General Surgery  
Box 1000

SHULER, ROBERT H. - Internal Medicine  
Box 438  
SPENCER, EDWARD D. - General Practice  
Box 1048

#### KODIAK

Population — 2,628  
Hospital Beds — 18 (Griffin Memorial-Catholic)  
JOHNSON, A. HOLMES - Semi-Retired  
Box 766  
JOHNSON, R. HOLMES - General Practice  
Box 766  
KEERS, J. BRUCE - General Practice  
Box 766

#### NOME

Population — 2,316  
Hospital Beds — 29 (Maynard-McDougall  
Memorial Hospital-Methodist)  
FENSTERMACHER, ROBERT E. - General  
Practice, Box 550  
SHADLER, JOHN A. - General Surgery

#### PALMER-WASILLA

Population — 1,852  
Hospital Beds — 25 (Valley Presbyterian)  
BAILEY, CLARENCE C. - General Practice  
Box J  
CUNNINGHAM, WALTER - General Practice  
HUME, VINCENT - General Practice  
Box 1833  
SKILLE, BOYD - General Practice

#### SEWARD

Population — 1,891  
Hospital Beds — 30 (Seward General Hospital)  
DEISHER, JOSEPH B. - General Practice  
Box 247  
GENTLES, ERNEST W. - General Practice  
Box 185

#### PETERSBURG

Population — 1,502  
Hospital Beds — 21 (Petersburg General Hospital)  
COON, DUANE A. - General Practice  
Box 1066  
SMITH, RUSSELL C. - General Practice  
Box 1054

#### BETHEL

Population — 1,258 mainly native  
Hospital Beds — 50 (U.S.P.H.S.)  
SCHIRMER, HARRIET JACKSON - General  
Practice, Box 286

#### HOMER

Population — 1,247  
Hospital Beds — 3 (Homer Hospital & Health Ctr.)  
FENGER, JOHN B. - General Practice  
Box 202

#### CORDOVA

Population — 1,128

Hospital Beds — 22 (Cordova Cmty. Hospital)  
ARNOLD, PATRICIA  
Box 218  
CHAPMAN, W. JOHN  
Box 218

#### CHUGIAK, BIRCHWOOD, EAGLE RIVER

Population — 801  
No hospital facilities  
SIMPSON, MARSHALL A. - General Practice  
Box 373, Eagle River

#### BIG DELTA

No population listed  
No hospital facilities  
ADAM, FELIX M. - General Practice

#### KENAI

Population — 779  
No hospital facilities  
BARR, ALLEN W. - General Practice

#### SOLDOTNA-STERLING

Population - 492  
No hospital facilities  
GAEDE, ELMER E. - General Practice  
Box 345, Soldotna  
ISAAK, PAUL G. - General Practice  
Box 345, Soldotna

#### DILLINGHAM-NEKNEK

Population - 673  
Hospital Beds — 42 (U.S.P.H.S.)  
LIBBY, JOHN E. - General Practice

#### SKAGWAY

Population — 659  
Hospital Beds — 8 (White Pass & Yukon Railway Hosp)  
SAMMAN, H. DAVID - General Practice  
Box 537

#### VALDEZ

Population — 555  
Hospital Beds - 16 (Valdez Hospital)  
DAVIS, CLARENCE - General Practice  
Box 265

#### GLENALLEN AREA

Population — 436  
Hospital Beds - 3 (Faith Hosp.-Central Alaska Mission)  
PINNEO, JAMES - General Practice  
SCHNEIDER, CHESTER I. - General Practice

#### HAINES

Population — 397  
No hospital facilities  
JONES, STANLEY - General Practice

#### SAND POINT

Population — 254  
No hospital facilities  
SANDBERG, CARL E. - General Practice  
Box 201

# Book Review

## The Health of the Inuit of North America — a Bibliography from the Earliest Times through 1990

Doctor Robert Fortune et. al. have compiled a 3/4 inch thick work containing references to virtually all the papers in the universe on the subject of "The Health of the Inuit of North America — a Bibliography from the Earliest Times through 1990."

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While this may not be a volume one would keep on one's night stand, it's nice to know it exists. Anything you ever wanted to know. . . .

Donald R. Rogers, M.D.  
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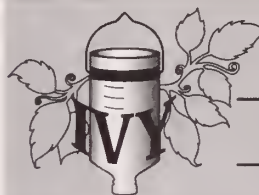
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(continued from page 106 - Lupus Erythematosus)

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(continued from page 114 — For the Record)

higher taxes to fund health reform, 63 percent do not agree with implementing global budgets, and 74 percent reject employer mandates.

Governor Hickel has proposed the creation of an Alaska Health Commission, in an effort to take a go-slow approach, recognizing that the health care system is not "universally broken in Alaska, but does require attention in specific areas." I agree with this approach and believe the Governor's proposal is a good starting point for Alaska. It addresses problems with administrative inefficiencies, data collection and malpractice reforms. The Commission's charge of reviewing reform proposals based on cost and efficacy for consumers, providers, and business will add balance to a debate that has been predominately one-sided to date.

Ultimately, my job is to ensure that federal reforms will allow Alaskans to choose what is best for us, while preserving and strengthening federal programs that are so critical to our state. Health reform is a great challenge, but not an insurmountable one. Let's make sure we get it right.

While it's acknowledged we have a crisis in health care, it is to a large degree, due to rapidly rising costs. Yet, at the same time, if you think health care costs are high now, wait until you see what they cost under the Clinton plan when they are "free."

# In Memoriam

## KEVIN BYRON PARK

Born: March 22, 1956 — Denver, Colorado  
Died: January 17, 1994 — Anchorage, Alaska  
Graduated High School: 1974 East High, Anchorage, Alaska  
BS General Studies 1978: Washington State University,  
Pullman, Washington  
MD 1982: University of Alaska/University of Washington  
Medical School, Seattle, Washington  
Internship 1983: Valley Medical Center, Fresno, California  
Residency Emergency Medicine 1985: Valley Medical  
Center, Fresno, California  
Partner: Alaska Emergency Medicine Associates

"When my group asked me to conduct a nationwide search for a superior Emergency physician I called the directors of the ten best residency programs in the country and asked for their top physician. Only one received universal superlatives. . . That was Kevin Park."

"Personally, Dr. Park is pleasant, extremely likable, highly dependable and a very interesting human being."

"His diverse outside interests help him keep the personal psychological balance with is so characteristic of him."

". . . Dr. Park is a truly remarkable human being and an excellent Emergency physician."

"Kevin epitomizes the personality of the compassionate physician."

On January 17th Kevin Park died. He died doing one of the many things he loved to do. While taking off on the Matanuska Glacier Kevin's engine seized and he died on impact. In that moment we lost a husband, father, son, brother, friend, physician and an Alaskan.

On the Friday after Kevin died many of us gathered to publicly and privately share experiences we each had enjoyed with this talented man. We heard how years ago he chose to stay in California rather than return to Alaska in order to pursue his lovely wife; how he built a log home doing much of the work himself and how that home became a repository for his tools. We closed our eyes and saw Kevin holding Connor high above his head both cheering wildly as UAA scored another goal and both with smiles that could conquer any heart. A nurse described Kevin sneaking Connor out of the nursery and bringing him down to the ER to share his proudest moment with his friends. "You only had to look at the pride in his face and the smile in his eyes to see the love



he had for his little boy, Connor." We learned Kevin was born in Denver and came to Alaska shortly afterwards. We discovered that he had followed his mother as a caring physician providing quality care to Alaskans and we relived Kevin telling about the many Alaskan adventures he had shared with his Dad. We met his brothers and sister and learned again what a source of strength he was to the family. We smiled with friends sharing experiences of how he helped them with one project or another; how he loaned this one his trailer and fixed the brakes on that one's car; and we heard a little girl tell how Kevin brought his light into her family's life with fireworks. Nurses admired his skills as a physician; his peers trusted his judgment and patients felt his gentle touch. He was an excellent physician and more than that he treated all people equally. One of his partners said, "He was good and kind, an independent thinker, and honest." Kevin hunted, fished, boated, flew, skied, played hockey — he lived the Alaskan life with a passion. More than that though he loved to share it with family and friends. One he taught to catch kings; another to whisper while hunting sheep and slogging up mountainsides; one to skate backwards; another to stalk moose and another the intricate workings of a Shopmate.

In life Kevin was described as "a truly remarkable human being and an excellent Emergency physician." In his death we learned that he was all of that and so very much more. The story of Kevin's life is rich and full and it is written. Neither our grief nor our tears can call him back to write another line.

Kevin, our husband, our father, our son, our brother, our doctor and our friend, we miss you. May flights of angels sing you to your rest.

John Hall, M.D.

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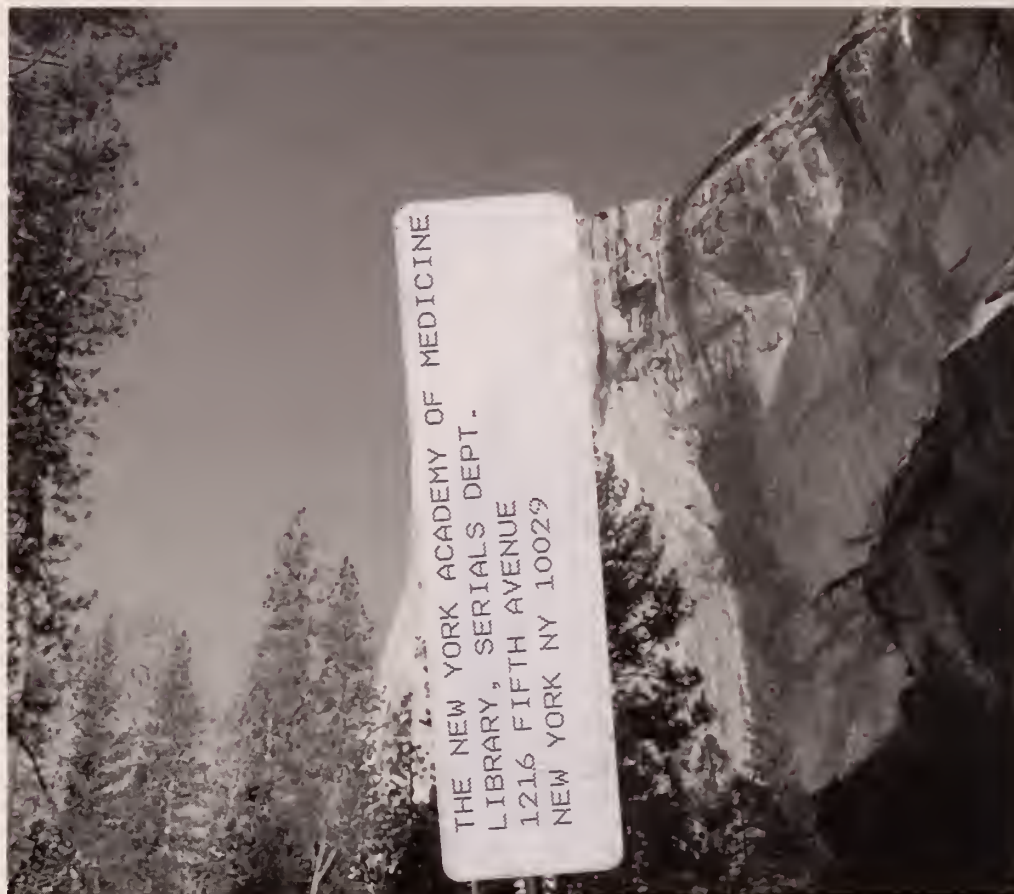
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Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalamic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

**Indications:** Yocon<sup>®</sup> is indicated as a sympatholytic and mydriatic. It may have activity as an aphrodisiac.

**Contraindications:** Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

**Warning:** Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

**Adverse Reactions:** Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral alpha-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.<sup>1,2</sup> Also dizziness, headache, skin flushing reported when used orally.<sup>1,3</sup>

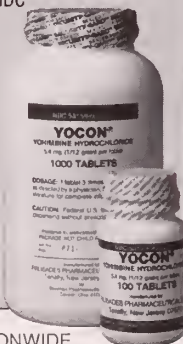
**Dosage and Administration:** Experimental dosage reported in treatment of erectile impotence.<sup>1,3,4</sup> 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to 1/2 tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.<sup>3</sup>

**How Supplied:** Oral tablets of Yocon<sup>®</sup> 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

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GOVERNOR



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STATE OF ALASKA  
OFFICE OF THE GOVERNOR  
JUNEAU

August 23, 1994

*Dear Fellow Alaskan:*

*The report included in this issue demonstrates Alaska's commitment to our circumpolar neighbors.*

*Health issues know no borders. The clinical exchanges between Alaska and the Russian Far East benefit not only our peoples, but the peoples of all northern regions.*

*On behalf of all Alaskans, I commend the dedicated health professionals who have made these exchanges a success. This worthy endeavor is a testimony to the Institute for Circumpolar Health Studies at the University of Alaska Anchorage.*

*With best regards.*

*Sincerely,*

A handwritten signature in dark ink, reading "Walter J. Hickel".

Walter J. Hickel  
Governor



# STATE OF ALASKA

## DEPARTMENT OF HEALTH AND SOCIAL SERVICES

OFFICE OF THE COMMISSIONER

WALTER J. HICKEL, GOVERNOR

MARGARET R. LOWE, COMMISSIONER

P.O. BOX 110601  
JUNEAU, ALASKA 99811-0601  
PHONE: (907) 465-3030

August 24, 1994

Dear Fellow Alaskans:

It has scarcely been a decade since the "ice curtain" between Alaska and the Russian Far East began to melt. The Institute for Circumpolar Health Studies at the University of Alaska Anchorage was among the first organizations to establish formal ties with our neighbors to the East.

The most recent fruit of the Institute's pioneering efforts is included in this issue of Alaska Medicine. As a participant in the Alaska/Russia Medical Exchange, I speak with enthusiasm about the program's benefits. Consultation and collaboration among health care professionals in the circumpolar region can and will lead to real improvements in all our peoples' lives.

The Department of Health and Social Services wishes to thank Alaska Medicine and all the dedicated professionals who have made the Russia/Alaska Medical Exchange a success.

Sincerely,

*Margaret R. Lowe*

Margaret R. Lowe, M.Ed., Ed.S.  
Commissioner



# UNIVERSITY OF ALASKA ANCHORAGE

OFFICE OF THE CHANCELLOR

3211 Providence Drive  
Anchorage, Alaska 99508-8060  
(907) 786-1437 — FAX (907) 786-6123  
AYCHANC

August 25, 1994

Dear Fellow Alaskans:

I am pleased to bring the reports of the most recent Alaska-Russia medical exchanges to your attention. These reports are the products of visitations and investigations of health and medical professionals from both sides of the Bering Sea working under the sponsorship of the University of Alaska Anchorage's Institute for Circumpolar Health Studies (ICHS).

Improved health care for northern peoples via education, research and service is an important goal of UAA. We are proud of the contributions of ICHS in pursuing this goal. This publication reports on the experiences and observations of Alaskan and Russian professionals who are striving to improve health care through increased communication. These reports are designed to improve our understanding of our health and health care commonalities and differences. I hope health and medical practitioners find it both informative and useful.

I want to extend a special appreciation to the Russian Ministries of Health for their continued participation in these exchanges; and all of the supporters of our Institute for Circumpolar Health Studies whose contributions have made this work possible. We look forward to future exchanges toward the end of improved health care for all northern peoples.

Sincerely,

A handwritten signature in black ink, reading "Edward Lee Gorsuch".

Edward Lee Gorsuch  
Chancellor

ELG:les



# UNIVERSITY OF ALASKA ANCHORAGE

## INSTITUTE FOR CIRCUMPOLAR HEALTH STUDIES



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August 22, 1994

Dear Alaskan and Russian Friends:

The Institute for Circumpolar Health Studies, University of Alaska Anchorage, is proud to present the third in a series of progress reports on recent activities in our Alaska/Russia Medical Exchange Project.

The following articles chronicle select reports of the seventeen working groups currently involved in research, training and professional exchange projects in the Russian Far East. Also included are select translated reports of Russian team members, many of whom have been participants in our projects since 1988.

We wish to express our gratitude to those people who have dedicated their talents and services toward better health and harmony for all people of the North. Our sincere appreciation to Governor Walter Hickel, Senator Jay Kerttula, Commissioner Margaret Lowe, Chancellor Lee Gorsuch, Dean Laura MacLachlan, Dr. Sergei Lisenko - Magadan Minister of Health, Mrs Nina Scherbak - Vice Governor of Magadan, the support staff of ICHS, our many volunteers and host families, and especially to the health professionals from Alaska and Russia who have joined hands across the borders in our common goal of healthy nations and peoples.

We pay tribute to their dedication and vision.

Sincerely,

Patricia L. Cochran  
Administrator

PLC:mah



# Alaska/Russian Far East Behavioral Health Projects: A Progress Report on Recent Activities

Bill Richards, M.D.<sup>(1)</sup>

Margaret Lowe, M.Ed., Ed.S.<sup>(2)</sup>

Mary Jane Starlings, MRC<sup>(3)</sup>

## ABSTRACT

A number of "behavioral health" exchange activities between Alaska and the Russian Far East took place during 1992-93. These included visits by a psychiatrist running one of the first "private polyclinics" in Magadan and by a narcologist to discuss the latest developments in alcohol services; a Behavioral Health Conference where Native health workers from Russia and Alaska shared experiences; beginning a multi-year epidemiology project; and a project to assess needs for a fetal alcohol syndrome (FAS) prevention program in Magadan.

## INTRODUCTION

Past Alaska Medicine articles (1,2) have described the beginnings of the Alaska - Magadan Behavioral Health Exchange Project in 1990, with follow-up activities in 1991.

This report provides an update on additional activities and exchange visits, with the pace of exchange activities increasing over that in earlier years, and with a broader base of involvement by State, Federal, and private sector service providers and researchers.

Private psychiatrist (Victor Bocharoff) from Magadan visited Alaska, to present information about the private fee-for-service polyclinics beginning to be possible under "perestroika".

The Chief of Narcology Services for the Magadan Region (Vladimir Lisenko) visited Alaska to present information about alcohol epidemiology and programs in Magadan and the surrounding settlements, as well as to study our services for FAS and high risk children of alcoholic families.

There was a Behavioral Health Conference at Meier Lake, where a team of Natives from the Magadan and Chukotka regions came to Alaska to exchange information with Alaska Natives about common problems and to share program strategies.

A National Science Foundation grant was awarded to a team in Alaska, working with counterparts in Moscow, to begin to study social transition in Native populations, comparing selected communities in Alaska and in the Russian Far East with respect to demographic changes, epidemiological trends, and patterns of change in families.

Another team from Alaska went to Magadan to work with Russian counterparts on a FAS data gathering project at a local Children's Home.

This report gives a brief summary of these various activities, some of which are reported in more detail elsewhere (3), since there are some common threads that may not be apparent from the individual reports. There is special attention on the work at the Children's Home in Magadan, since that project provides a model for clinical "behavioral health" research exchange projects that should be of interest to other Alaskan health professionals.

## BRIEF SUMMARIES OF EXCHANGE ACTIVITIES IN "BEHAVIORAL HEALTH"

### PRIVATE PRACTICE IN MAGADAN:

A visit by private psychiatrist Victor Bocharoff, M.D., from Magadan to Alaska, was organized early in 1992. Bill Richards, M.D. from Alaska Area Native Health Service, and Aron Wolf, M.D. from the Langdon Psychiatric Clinic, arranged for the visit. Dr. Bocharoff brought a video describing his private "polyclinic" in Magadan, and gave presentations at a number of locations in Alaska to various health organizations about his work.

Before "perestroika", the health system in Russia was basically all a "socialized medicine" system, with no private practice. Under the new laws, it is possible for doctors to have private practices for about 4 hours per

- 
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  3. Executive Director, ARCA, 2211-A Area Drive, Anchorage, Alaska 99508

day, plus weekends, although they have to continue with public practice for the rest of the work week.

Dr. Bocharoff has organized a whole "polyclinic", which includes psychiatrists, but also a number of other medical specialties - dentists, plastic surgeons, etc. and some specialists not so familiar in most of our clinics - acupuncturist, massage therapists and hypnotists among others.

He has worked out an interesting way of funding this clinic by convincing local factories to sign up for "pre-paid" coverage plans for groups of employees. The instability in the Russian economic system and high tax rates, were making business conditions very difficult, so he was diversifying the clinic's activities with a variety of "joint venture" projects extending outside of health into economic development activities and import-export arrangements.

Further write-ups of the information he presented are available in mimeo form, and are being submitted for publication (4).



Commissioner Lowe and Dr. Lisenko visit Ola Children's Home

attendance might be used to discriminate against them in the job market. There is considerable interest in finding Russian-speaking members of AA in the United States, who could help the local Russian groups develop their programs and considerable discussion about whether AA should be practiced in standard ways or would need to be adapted to fit Russian culture.

Compared to Alaska programs, there is much less in Magadan in the way of prevention and early intervention programs, little in the way of programs for FAS, school-based or community education programs, teen clinics, etc. The number of "narcology beds" in the Region has been cut from 400 to 200 in the space of a year because of economic problems, so programs are currently going through a difficult period of retrenchment, with doctors looking for other work or moving to other parts of Russia.

Additional details on the information presented by Dr. Lisenko is available and is being submitted for publication(5).

## ALCOHOL SERVICES IN MAGADAN:

A visit by Vladimir Lisenko, M.D., Chief of Narcology Service for the Magadan Region, took place in the Spring of 1992. This trip was organized by the State of Alaska Department of Health and Human Services, with assistance from Bill Richards at Alaska Area Native Health Service, and the Institute for Circumpolar Health Studies. Dr. Lisenko presented information at various locations in Alaska about the current state of alcohol epidemiology and services in the Magadan Region.

Magadan has high reported rates of alcohol abuse. Efforts in 1985-86 to restrict alcohol sales, under Gorbachev's "Alcohol Reform Law" program, were only successful for a short time. There was growth of "home brew" availability, so that vodka sales went down but people drank home brew instead. Now that vodka sales are less restricted by the government again and the home brew is also very available, people seem to be drinking more than ever.

There were some interesting developments that Dr. Lisenko reported on, such as the emergence of Alcoholics Anonymous (AA) groups in Magadan as well as in surrounding villages. Until recently, "secret societies" were prohibited in Russia, people attending AA meetings are still very concerned that information about their

## BEHAVIORAL HEALTH CONFERENCE AT MEIER LAKE:

This conference was organized by State of Alaska's Department of Health and Social Services - Division of Mental Health and Developmental Disabilities (Susan Soule and Margaret Lowe) with the assistance of the Institute for Circumpolar Health Studies, and was a first of its kind.

The conference allowed Native health professionals - involved with behavioral health issues in the Russian Far East - to exchange information directly with Natives involved with Alaska programs. A "retreat" setting was chosen to encourage maximum sharing of information with a small group. Approximately 40 people attended.

There has not previously been a large degree of involvement by Natives in the Russian Far East. Especially not those who live in the more rural settlements in regard to planning and managing their own health programs. Support for local Native associations has decreased considerably as the Communist Party has lost power, and the Native associations that do exist now have small staffs, low budgets, and are focused largely on land reform and economic issues rather than health or behavioral health.

The experiences of Alaska Natives was a topic of many very interesting discussions, where programs are run



under a "self-determination" philosophy, with considerable local input into program direction, regional health corporations, village-specific programs, etc.

Much information on the problems and programs of Natives in the Russian Far East was presented, some of which was in written form and is still being translated. Many of the problems were similar to those of Alaska Natives, especially the alcohol-related problems. Some interesting approaches were presented. They involved "mobile medical teams" providing services to remote nomadic reindeer herders who traveled to the herders by helicopter, and then lived in tents while carrying out their clinic duties.

Summary reports and resolutions from this conference are available (6). Plans for a follow-up conference, possibly in Khabarovsk, are being explored.

One of the participants at this conference, Dr. Larissa Abryutina, a Chukchi physician from Bilibino, was able to return later in the year during the Alaska Federation of Natives (AFN) annual convention. She brought a number of fascinating videos and slides showing life and health care for the reindeer herders in her region. There are copies available of this material, and the "sound tracks" on the videos are in the process of being translated into English (7).

#### NATIONAL SCIENCE FOUNDATION "SOCIAL TRANSITION" PROJECT:

This project was funded midway through 1992.

Funding is for four years, with principal investigators Steve McNabb, Ph.D. (Social Research Institute - Anchorage), Bill Richards, M.D. (Alaska Area Native Health Service - Anchorage), and Alexander Pika (epidemiologist and anthropologist at Institute of Demographic Studies - Moscow).

The project involves a multi-disciplinary team, with anthropologists, demographers, epidemiologists, ecologists, and psychiatrists, both Russian and Alaskan, and will involve field work in selected communities in the Russian Far East and in Alaska. Tribal organizations are involved in the research design. There have been major shifts, both for Russian Natives as well as Alaska Natives, in demographic and epidemiologic patterns over the past 50 years. The project will describe these in considerable detail using a variety of data collection methods, i.e.

surveys, interviews, ethnographies, participant observation, analysis of secondary data, etc. The focus will be on impact to Native families, i.e. size and composition, changes in roles, child development patterns, and on practical applications of the information collected for uses in program development and setting of health policy.

There have been exchange visits between the Alaska and the Russian teams to work on the project's methodology and instrumentation. Some of the preliminary work has been presented at a recent "Arctic Social Sciences" Conference in Quebec (8-10).

Susan Soule from State of Alaska's Division of Mental Health, and David Marshall, a private researcher focusing on suicide patterns, also have been considering possible research on suicidal behavior among Natives in the Russian Far East compared to Alaska Natives.

#### ALASKA - MAGADAN "FAS" PROJECT:

The exchange activities outlined above provide a

general picture of the range of projects currently under development.

This section of the report describes in greater detail one project that has been evolving for several years. It illustrates some of the methodology that can be used in "behavioral health" applied clinical research.

The Alaskans doing the main work on the project consist of a State-Federal-University-private team, consisting of:

- Margaret Lowe, Commissioner of the Alaska Department of Health and Social Services
- Dr. Bill Richards, Director, Division of Behavioral Health, Alaska Area Native Health Service
- Mary Jane Starling Michael, Executive Director, ARCA
- Patricia Cochran, Administrator of the Institute for Circumpolar Health Studies, UAA.

Involvement from the Magadan side includes:

- Dr. Sergei Lisenko, Magadan Region Minister of Health
- Dr. Vladimir Lisenko, Chief of Narcology Services
- Dr. Yuri Uelkhin, Chief of Pediatrics, Magadan Regional Hospital.



Infant at Children's Internat facility in Magadan



A memo of agreement was negotiated for the project between the Magadan Ministry of Health and the involved Alaskan organizations. A five-year plan also was developed.

The project's main focus is on problems of high risk children being raised in alcoholic families, with special attention to Native families. There are a number of special boarding homes or orphanages in the Magadan Region. These places take care of children with disabilities and "refusal children", those children whose parents do not want to care for them. A disproportionately high number of the children in such homes are Natives.

The project staff modified a protocol already in use in Alaska to check for FAS. It involved standardized examinations on the children residing in one of the homes mentioned. It included questions on the mother's alcohol intake during the pregnancy, developmental information on the child and physical indicators that might be associated with FAS, i.e., small head circumference, wide-set eyes, characteristic facies, short height and low weight and other congenital anomalies. The protocol was translated into Russian, and then "back-translated" into English to insure satisfactory interpretation.

A team with both Russian and Alaskan physicians simultaneously saw 22 children. This was to insure agreement on how the protocol questions would be presented. Photographs were taken of the children examined. Medical records, school records and psychological assessment reports were also reviewed.

Some members of the Alaska team gave in-service training sessions to the staff at the Children's Home. Since many of the approaches used in Alaska (infant stimulation, working with children using stronger sensory modalities and specialized functional assessments) were not being used by the Russian staff, visits to outlying facilities and rural settlements were made to discuss ways of extending services beyond the children's home.

Videos were also made depicting conditions within the Children's Home. A brief summary video is now available (11).

A survey seeking information known about the effects of drinking during the pregnancy, attitudes toward family planning, age of first sexual contact, smoking, and other "behavioral risk factors", was distributed to sample the

population's responses from the region.

Some of the preliminary findings of this work included:

1. Of the 22 children examined, relatively few had evidence strongly suggesting FAS.
2. Most of the children examined, however, had been severely impacted by alcohol abuse of their parents. The charts summarize some of this information. One child had been thrown against the wall by alcoholic parents, leading to a skull fracture and quadriplegia. Another child was from a family where the mother had killed the father during an alcoholic argument, and the baby was then born in jail. A majority of the children had one or both parents involved in severe drinking.
3. One would expect more FAS children than were actually found. The relatively low numbers may be

because these children were in remote settlements or in special school programs, rather than in the Children's Homes; or because the high abortion and infant mortality rates may affect results.

4. A number of other problems were found among the children at the Children's Home, who received minimal treatment compared to what would be standard in the U.S. These included children with: Down's

syndrome, who were hospitalized with mini-mum special education rather than being main-streamed as in the U.S.; spina bida; seizure problems; hydrocephalies, where no shunting was available; children with cleft palates or dental or orthopedic problems where no treatment had been given; and children with missing limbs without prostheses, wheel chairs or other special assistive devices.

5. Possible "behavioral health risk" diagnoses are listed in the attached chart.

A visit by the Russian pediatrician to Alaska is planned as a next step. The intent is to gather additional information about FAS, as well as how other childhood disabilities are handled in Alaska.

Grant support is being sought from a variety of U.S. sources to carry out additional parts to this project, since it currently is very difficult for the Russians to come up with monies for travel and project support costs. Also being considered is an epidemiological project which



Children's Internat facility

would study high risk children being raised in alcoholic families, with a focus on the Native families in the remote settlements, and on a variety of other alcohol-related problems extending beyond fetal alcohol syndrome.

## SUMMARY

All of the projects outlined above have some common features. They are all set up as "exchange" projects. The approach is one of a two-way exchange of ideas and mutual goal setting, rather than a unilateral "technology transfer" to provide technical assistance or resources from a paternalistic perspective.

The participants are not completely equal at this time for any of these projects. This is largely because of the instability and deteriorating economic conditions in Russia, and the relative isolation that has been experienced by health professionals in the Russian Far East until very recently. Alaskans have, therefore, had to be active in finding funding, and in planning and organizing some of the exchange activities. However, all of these projects basically are aimed in building up two-way exchanges of a truly collaborative nature.

The projects focus on Native populations, and on comparing Native groups in the Russian Far East with those in Alaska. Arctic conditions are similar in both locations, and there are many similar problems, with very difficult solutions. This makes joint projects very interesting, since assumptions and points taken for granted by one side may be questioned by the other side. This leads to opportunities for fresh ideas for ways health programs can function.

The project with FAS has a number of features that have been learned by trial and error. These features include:

- The need for a memo of agreement with the key organizations involved;
- A "five-year plan";
- Ways to develop survey instruments and "back-translate" them to check on accuracy of the translation;
- Use of multi-disciplinary teams which include pediatricians, narcologists, experts in special education, etc.;
- The use of a "training and development" model

where research is combined with in-service training;

- Public opinion surveys to gather information for prevention and education efforts;
- Starting with an institutional base, which the Russians are used to, and then attempting to extend out to rural settlements;
- Starting with a condition where there are physical markers, such as FAS, to avoid the difficulties in doing behavioral health studies on conditions where diagnosis can be much more subjective, etc.

Funding is being sought from National U.S. organizations that would not otherwise be available to Alaska, to build capacity both here and in Russia.

Relatively "low cost", "low technology", practical applications of methods were used, such as using available toys to demonstrate the principles of infant stimulation, rather than high tech devices. Use of volunteers and voluntary contributions have also been a part of this effort, including encouraging contributions of wheel chairs and other equipment and donation of staff time at the Children's Home, etc. There has been interest in this model from other



Child at Internat

parts of the Russian Far East, including Khabarovsk and Provideniya,. Future projects may be involved at these locations.

## PUBLIC OPINION SURVEY:

- Carried out in the Magadan Region, February - April, 1992
- Sampled:

223	Parents of children less then 7 years old
219	Parents of children in 1st and 3rd grade
364	Parents of children in 7th and 11th grade
436	Students in vocational schools
286	Students in institutes
265	People getting married
120	People getting divorced

There were questions on attitudes about family planning, age of first sexual contact, knowledge about FAS, smoking, etc.

## Statistics of responses to some questions:

1. Have you ever heard in your life about FAS?	Yes No		5. If the parents don't drink at all, will it be good for children?	Yes No Maybe Don't Know			
Married	43.78%	56.22%	Married	66.28%	14.33%	2.26%	17.13%
Divorced	69.11%	30.89%	Divorced	80.18%	9.23%	1.02%	9.57%
2. Is FAS a defect of birth?	Yes No Hard to say			6. To help the mothers of children not to drink, the father shouldn't drink either during the period of the pregnancy.	Yes No Don't Know		
Married	30.94%	15.44%	53.58%	Married	69.54%	18.11%	14.35%
Divorced	63.22%	12.02%	24.76%	Divorced	81.04%	10.2%	8.76%
3. If children have FAS, will they always have low development?	Yes No Maybe Don't Know				7. Do you use birth control?	Yes No	
Married	19.24%	10.56%	24.92%	45.28%	Married	58%	42%
Divorced	51.09%	9.37%	17.36%	22.18%	Divorced	83%	17%
4. Can you prevent FAS?	Yes No Don't Know			8. Is abortion normal as away to prevent pregnancy?	Yes No Hard to say		
Married	46.42%	2.64%	50.94%	Married	9.82%	74.71%	15.47%
Divorced	36.73%	25.04%	38.23%	Divorced	15.47%	80.35%	4.18%

## DIAGNOSIS OF PATIENTS SEEN

Case	Sex	Age	Diagnosis	Nationality
1	F	12	Oligophrenia - idiot; microcephaly	Russian
2	M	12	Oligophrenia - idiot; microcephaly	Russian
3	F	11	Oligophrenia - idiot; cleft palate; clinodactyly; leg contracture; possible FAS	Russian
4	F	12	Oligophrenia - idiot; very widest eyes; ears deformed; bad teeth; possible FAS	Nebe
5	M	8	Oligophrenia - idiot; childhood cerebral palsy	Russian
6	M	6	Oligophrenia - imbecile; paraparesis; microcephaly; cryptorchidism; visual atrophy	Yakut
7	M	19	Early childhood schizophrenia	Ukrainian
8	M	10	Oligophrenia - idiot; Downs; very flat upper lip	Russian
9	M	6	Oligophrenia - idiot; Downs	Yakutsk
10	F	8	Oligophrenia - idiot; Epilepsy; possible FAS from facies, head measurements	Russian
11	F	10	Oligophrenia - idiot; quadriplegia; microcephalic; widest eyes; upturned nose; possible FAS	Chukchi
12	F	6	Oligophrenia - possible FAS by physical appearance, facies	Russian
13	M	6	Oligophrenia - imbecile; "central paralysis of children"	Russian
14	F	6	Oligophrenia - idiot; paraparesis; possible FAS by physical appearance	Russian
15	M	1 1/2	Epilepsy; developmental delays; possible FAS by physical appearance	Russian
16	M	1 1/2	Show development; possible FAS	Russian
17	F	4	Skull fracture secondary to child abuse	Russian
18	F	2	Encephalopathy; low development; possible FAS by appearance	Russian
19	F	8 mos.	Cleft lip; low development; possible FAS by appearance	Chukchi
20	F	3	Delayed development; 7 debile or oligophrenic	Chukchi
21	M	2	Low development; encephalopathy; possible FAS	Orachi
22	M	2 3/4	Encephalopathy; birth injury to head; possible FAS	Russian

Russian diagnostic system speaks of "debiles" (able to work, usually go to special schools), "imbeciles" (moderately retarded; can be taught self care skills with special training), and "idiots" (severely retarded; can't be taught to feed or dress themselves).



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Dance at Children's Internat



Dr. Lisenko and Dr. Richards at the Magadan Narcology Clinic

# Russian Oral Health Initiative — Magadan Region And Seymchan

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## INTRODUCTION

Americans have called it Siberia. Siberia, however, lies west of Russia's Pacific Rim regions known collectively as the Far East. In 1989 initial contact was established between dental professionals in the Far East and Alaska through exchanges sponsored by such organizations as the Institute for Circumpolar Health Studies and Rotary International. The Russians sincere desire to improve their dental care, their interest in American products and technology, and their friendliness and warmth have spawned a continuation of exchanges and friendships with their American colleagues.

In August of 1992 the Health Minister of the Region of Magadan and several regional doctors hosted three American dental professionals, two of whom had participated in earlier exchanges. The voluntary expedition was funded by the three American participants and the Magadan Executive Committee of Health. Instruments and supplies were donated by several dental supply firms and manufacturers. The purpose of the 1992 exchange was twofold: 1) to assist the Magadan Health Ministers and Regional Health Administrators in developing a long-term preventive dental health plan for the Region, 2) to share technical knowledge and demonstrate clinical preventive techniques.

## DEMOGRAPHICS

Magadan, population 150,000, is the largest city of the Region bearing the same name. The Lenin Highway connects the Baltic Sea and St. Petersburg with Magadan and the Pacific Ocean and is the longest road in the world.

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Magadan has no railway links to the rest of Russia. Both Aeroflot and Alaska Airlines provide air service to Anchorage. Business and political connections exist with Japan, Korea and China. Major trade items are the horns and meat from reindeer raised on farms throughout the Magadan Region. Horns are in great demand by Asians, who use them as aphrodisiacs, and reindeer meat is made into a popular sausage.

Some clinical activities occurred in Magadan, but the main clinical exchange was conducted in Seymchan, a smaller city located on the Kolyma River 300 miles due north of Magadan. Seymchan lies at approximately the same longitude as Fairbanks, Alaska, and resembles Fairbanks in both climate and geography. Helicopters and AN-2 biplanes provide air transportation in the

Region. A gravel road which connects Magadan and Seymchan is used mainly by mining trucks operating out of the area's gold, tin and mercury mines. Over 20 gulags, hard labor camps started by Stalin in the 1940s, dotted the area and many of the current 15,000 population are former prisoners or their descendants. Included too are people from throughout the former Soviet Union who have been relocated

to the Far East in return for better salaries and benefits. Products from local dairies, fishing, reindeer farms, greenhouses and gardens provide food for much of the Magadan Region.

## DENTAL CARE SYSTEM

Three modes of dental care delivery seem to be operating in the Far East. State funded school-based clinics and community polyclinics serve most of the people in Magadan and Seymchan. In Seymchan children routinely receive examinations and treatment every two years in the community polyclinic. Also, throughout the



Dental team at a Magadan dental clinic



region, clinics associated with specific facilities or populations, such as airline and port personnel and mining cooperatives, provide dental care. Magadan Regional Health Directors are working currently on a form of health insurance coverage which would split costs between the patient and cooperatives. It would combine the free market economy and government benefits. Initial plans call generally for complete coverage up to age 14 with a focus on prevention. Third, a limited amount of private services are rendered. Russia is divided into regions or 'states' called oblasts. Each oblast is served by a Chief Stomatologist who is responsible for the placement and supervision of dentists, acquisition of equipment and supplies and program planning for state-funded clinics.

The Chief Stomatologist works directly under the regional Deputy Minister of Health who is responsible for all clinical treatment modalities. The region's Health Minister or Chief Doctor is responsible for all health care under his/her jurisdiction. Russians say that officially the only Health Ministers that exist in Russia are located in Moscow while the regional ministers call themselves - Chief Doctors'. The interchanging of these titles may offer insight into relationships between Moscow and the Far East. There is a saying, "God is high in the sky and Moscow is far away".

The Chief Stomatologist of Magadan is a progressive woman who appreciates the need for modern technology and equipment. She values dental prevention, has enthusiastically elicited support from her colleagues, and is solidly supported by the Chief Doctor/Administrator of the Magadan Region. She is a key figure in the organization and success of dental exchanges between Alaska and Magadan and requested that this exchange be focused on preventive education and integration of sealant placement in public health practice. Also, periodontal awareness and the dental hygienist's role in delivery of preventive services and in raising the quality of oral health care were new ideas to share.

## THE EXCHANGE - Planning Meetings

An initial meeting was held in Magadan by the Health Ministers and Administrators to solicit input for a preventive oral health initiative they were developing for the entire region. This formalized plan would serve to initiate programs focused on prevention of oral diseases and

to provide direction for future exchanges. A preliminary outline was developed and arrangements made to meet and finalize the draft near the end of the visit. This provided the American participants time for observation and the opportunity to develop more insight.

A tour of the Magadan School of Nursing was arranged by the Director who is keenly interested in developing a dental hygiene program. He had been in communication with one of the exchange participants regarding a program and pursued more definitive information with her. The school currently educates nurses who specialize in dentistry. However, compared to duties of dental assistants in this country, these dental nurses are utilized minimally in the dental operatory and typically do not participate in patient care. In 1990 the school opened a dental laboratory technician program and to date has graduated two classes. Recently the school obtained two new dental units made in Czechoslovakia and now has a stomatologist on the faculty. Both promising developments for the inclusion of a dental hygiene program.

## THE EXCHANGE - Clinical Activities in Seymchan

Seymchan was selected as the site for the clinical exchange following an epidemiological dental survey conducted in Seymchan in 1991 by Alaska Public Health and World Health Organization dental officials together with health officials from Moscow. The Chief Doctor of Seymchan and his administrator served as hosts for the seven-day visit. The Chief Doctor is an innovative individual with foresight. His hospital has the first computer database in the Region, the first-public health nurse' position has been established in the children's



Dr. Arneson watching dental procedure

ward of his hospital, and he strongly supports establishing preventive dental health programs and services.

First, the Americans were welcomed into local kindergartens to give presentations on prevention, teach tooth brushing and screen children 5-7 years of age for placement of sealants or preventive resin restorations (PRR). Children so identified were then taken to the local polyclinic where placement of sealants and PRRs was demonstrated. Russian stomatologists from several clinics assisted. Then switching roles, the Russian clinicians placed sealants. After their proficiency increased, they assisted each other while the Americans gave suggestions and answered questions. The technique sensitive



procedures of acid etching and resin adhesion were explained and practiced in detail. Rubber dam utilization and composite/PRR placement were thoroughly reviewed and demonstrated.

Periodontal sealing was demonstrated and performed on a few adult patients. Little or no periodontal assessment or treatment is being performed currently but participating stomatologists expressed strong interest and one eagerly participated as a patient. All expressed appreciation for the sharing of knowledge and techniques and seemed especially interested in improving the children's dental health. Appreciation was expressed throughout the visit and formally at a luncheon featuring local specialties prepared by the Russian participants.

Following is a list of activities and services performed in Magadan and Seymchan:

- 150 children screened and given oral hygiene instructions
- 121 sealants placed in children's teeth
- 11 PRR/composite restorations placed
- 5 full-mouth sealings completed
- 10 patient consultations provided for adults and children
- 12 stomatologists trained

A record of children who currently required restorations or would need sealants when their six-year molars erupted further was provided to the children's stomatologists. Official meetings were held with area school teachers, nurses and physicians to share preventive information and philosophy and to explain the clinical procedures performed on the children. It was learned that some teachers had been having children brush in the classroom since they received toothbrushes and instructions from the 1991 American survey team.

## ORAL HEALTH INITIATIVE

A final meeting with the Health Ministers in Magadan resulted in the formulation of a plan for writing the oral health initiative. All agreed that support for the initiative should be solicited from all of the various professional groups from Alaska who had participated in recent exchanges. This would enhance the coordination and success of future professional exchanges and give more credence to the initiative when the Governor of the Region presented it to Moscow for approval and acquisitions of funds.

The four key points developed by the Russians for the initiative are:

1. Oral health education and preventive restorations (includes methods to incorporate fluoride for caries prevention)

2. Education facilities and programs to train dental hygienists and dental assistants
3. Education and skills training of current and future stomatologists in recent technological advances
4. Purchasing of dental equipment needed to perform new techniques and obtain optimal results

## SOCIAL EVENTS

The trip also included many memorable social events with the wonderful people of Seymchan and Magadan. The Americans were privileged to be invited to a wedding and the festive dinner celebration which followed. A scenic boat ride down the Kolyma River provided an enjoyable outing to a summer cabin where hot berry punch and fish soup, a local specialty prepared over an open fire, were served as part of the hospitality. Some of the most memorable times were simply having tea and conversation with the new Russian friends.

## REVELATIONS AND RECOMMENDATIONS

This trip exposed/confirmed some challenges for the success of the oral health initiative and for future professional exchanges.

1. Existing dental equipment provided air which was not moisture or oil free. High volume evacuation doesn't exist so suction devices aren't operable. The ubiquitous cuspidor served as the primary form of moisture control and waste receptacle. Dental units were not conducive for auxiliary utilization and often did not function fully. It appears that equipment repair specialists are not available. Portable American equipment was used for this exchange, but before further clinical exchanges are practical new equipment must be procured.
2. A reliable supply of materials for sealants, dentin bonding composites and glass ionomer restorations along with education in the use and chemistry of these materials are needed. Already some Russian plants which have affiliations with American companies have started manufacturing modern dental materials.
3. Fear of all dental care exists. Anesthetic usage is reserved for dental extractions. The first exposure to dental care is often for a toothache. The excellence and enthusiasm of the interpreter for this exchange made a crucial difference in the children's acceptance and reaction to care. In the classrooms she used Russian fairy tale 'creatures' to explain bacteria and how they cause tooth decay. Then she cleverly announced that the dental drill was broken so no drilling would be done, but that invisible 'blankets' were going to be placed on their teeth to prevent cavities. Also, she helped alleviate anxiety of many

children when they arrived at the clinic. The painless placement of sealants was well received by the children and impressed the adults. Smiles abounded when stickers and toothpaste were distributed following the procedures.

4. Hepatitis and tuberculosis are major health concerns in this area. Infection control protocols need to be followed and stressed by visiting clinicians; including the fact that disposable needles are not reusable. Sterilizers are available but should be tested for effectiveness. Alcohol is used for disinfection but seemed to be in short supply.
5. Education programs in the utilization of dental auxiliaries and in preventive dentistry and periodontics are critically needed. While great interest exists in ceramic technology, fundamental principles necessary for this type of dentistry and more advanced prosthodontic procedures will require in-depth, step-by-step training. A dental hygiene program would provide critically needed dental health educators, preventive care providers and clinicians capable of rendering initial periodontal services.

## CONCLUSION

An environment exists for implementation of a model dental prevention program in Scymchan and Magadan. It has been started.

- A. A plan to train Russian stomatologists in dental hygiene techniques and procedures in preparation for a dental hygiene program in the Magadan School of Nursing is being formulated. The Russians hope to have the program in operation within two years.
- B. The oral health initiative developed during this trip was taken back to Alaska and was given almost unanimous support and signed by the Alaskan professional groups who have been involved in many dental health exchanges over the last several years. It was returned to Magadan officials in October and then presented to the Health Ministers in Moscow. This oral health initiative was instrumental in obtaining a significant sum of hard currency that has been allocated to the Magadan Region for improvement in oral health care. Subsequently, in April 1993, the Chief Stomatologist of Magadan came to the United States to explore the purchase of dental units and supplies upon receipt of promised funds.
- C. As of fall 1993 portable equipment has been purchased and is currently being utilized in some of the clinics in the Magadan region, with emphasis being placed on utilizing for placement of sealants for children.
- D. Several exchanges besides this effort are occurring with other dental professionals in Alaska. It is hoped that a line of communication can remain open and

combined efforts to a common cause will expedite even greater changes.

- E. The initiative is currently dependent on the politics and economics of the region. As is apparent in the media, this is a very unstable situation. It is hoped this will stabilize and progress will continue.

Continuation of professional exchanges between Russia and America and access to materials and equipment are key ingredients toward the success of this innovative and remarkable initiative.

## ACKNOWLEDGMENTS

The authors extend a warm thanks to our gracious Russian hosts for their excellent planning, accommodations and friendliness. Appreciation from all who participated and benefited from this exchange is extended to the many individuals, organizations, and companies who generously donated materials or loaned equipment for this exchange.

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# Alaska/Russian Far East Women's Health and Family Planning

Russel J. Thomsen, M.D.<sup>(1)</sup>

Since 1988 I have had five opportunities to visit health care facilities throughout the former Soviet Union including the Russian Far East, and have been able to compare health care, family planning and women's health issues. During my trip to the Magadan Oblast in July and August of 1992, I observed and worked in regional hospitals and clinics, as well as smaller



Staff of Magadan Gynecological Hospital. Dr. Yeliseikin, center; Dr. Ulykin, far right; and Dr. thomsen, fourth from right

district and village facilities.

The Magadan Maternity Hospital, which also contains the Magadan Gynecological Clinic and Hospital, and the Maternity Polyclinic, is the principal facility in the City of Magadan that I concentrated my time on. Dr. Stanislav Yeliseikin, the Head Physician of the Magadan Maternity Hospital, explained that while this is generally the English name for the institution, it is more correctly a "Women's Hospital" serving Magadan. It contains a general out-patient gynecology clinic, administrative offices, the city's gynecological surgical area, inpatient gynecological ward and an extremely busy abortion service.

With Dr. Yeliseikin, I performed an abdominal supra-cervical hysterectomy, for a "14 week" myoma. The procedure was done using general intubation anesthesia, using reusable gloves, a resharpened knife and no electrocautery. I observed good surgical technique from my Russian colleagues.

I later saw five abortions being done in the span of twenty minutes. These were done in one room with two gynecological tables and two benches for patient re-

covery. A typical technique was one waiting awake patient on one of the benches, observing everything. One patient was on a table recovering from the IV ketamine. The second table was being used for the abortion procedure. The patient is anaesthetized by a direct IV injection of ketamine by syringe, rather than from a running IV line. The patient rapidly goes under narcosis. The cervix is grasped with a tenaculum, with a cleaning solution being applied. Two large dilators were used quickly, followed by a very firm (even rough) curettage with the assistant holding the tissue pan under the table. A suction machine is in the room but apparently seldom used. The two to three minute procedure is followed by the placement of a hot water bottle on the patient's abdomen. This is followed by a few minutes of recovery, then the patient is walked to a post-abortion ward.

The abortions that day were being performed by Dr. Tamara Sharovatyh, who has been working in Magadan for thirty years, and averages 3000 abortions per year. I commented to the team on its "efficiency", the need for modern birth control and sterilization to replace this quantity of abortions, and the change in blood handling techniques which will come as concern about AIDS spreads to the Russian Far East. I inserted six NORPLANT



NORPLANT recipient and her family in the village of Ola, Magadan

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Dr. Yeliseikin and Dr. Thomsen perform hystrectomy

systems under observation by about twenty physicians and staff.

The Clinic of Marriage and the Family was housed in its own building and organized separate from the Maternity Hospital and associated institutions. It is funded by the Magadan Ministry of Health and the Magadan Executive Committee. Comparable to a family planning clinic in the U.S., it is a comprehensive clinic for sexual, family planning and abortion issues. Infertility workups are done here, including artificial insemination, tubal insufflations and sperm counts. It has no ultrasound or laparoscopic abilities. It works with male and female sexual dysfunction, including the use of electrical stimulation, and suction catheter for transurethral stimulation and drainage of the prostate in prostatitis. Dr. Eugeni Kuznetsov, Chief Physician of the Clinic, claims a high success rate, and that it was one of only five centers in Russia to use such a catheter. I was also shown a vacuum device for the treatment of male impotence. The clinic also does counseling and a variety of acupuncture treatments for a multitude of complaints, something I have seen in other gynecological centers in the former Soviet Union.

While at the Clinic, I demonstrated more NORPLANT insertions, and discussed techniques and followup. I obtained a sample of the Organon Multiload Cu-375 IUD that included Russian language packaging - it was the first time I had seen it in Russia, and it now seemed generally available. I left several TCu-380-A IUDs at the Clinic.

I traveled to the small district hospital in the town of Ola, some thirty kilometers northeast of Magadan, which serves a population of about 15,000 people. The

hospital has about 250 deliveries per year, with a staff of four gynecologists. There were no ultrasound, monitors or doptones. They did have some Russian Multiload or Copper T IUDs, but no birth control pills. They routinely use abortions, of course, for most family planning. I gave a lecture to about thirty health care providers, and did more NORPLANT insertion demonstrations.

One hundred-ninety kilometers northwest of Magadan is the town of Ust-Omchug, which also has a district hospital serving about 15,000 people. Dark and with minimal supplies, it is run by motivated professionals. After presenting a lecture, I did a teaching NORPLANT insertion on the hospital's chief gynecologist, and left supplies with the staff.

While I was in Magadan, I met with a three member delegation from Project Hope, which was surveying medical aid needs across the former Soviet Union. I mentioned that for all practical purposes this area had not been a recipient of U.S. humanitarian aid, and was furthest from the "Moscow pipeline", and in many ways medically the most destitute.

Prior to my departure from Magadan, I met with a number of the doctors I had worked with in the city of Magadan, and Dr. Sergei Lisenko, Minister of Health for the Magadan Oblast, and his deputy, Dr. Alla Nikitina. The nature of our long discussion can be summarized as follows:

1. Dr. Lisenko expressed appreciation for the personal approach I made to both professionals and patients during my visits to the various institutions.
2. There was clear discussion of the Magadan Oblast's funding of a cooperative agreement with Alaska in the area of maternal-child health and family planning. This would be a working relationship including training on both sides.



Dr. Thomsen demonstrates NORPLANT insertion at the Magadan Gynecological Hospital.

# Alaska/Magadan Radiology Project Update

William Cox, MD <sup>(1)</sup>

My relationship with the Radiology Department of the Magadan Regional Hospital began in early 1990 when the Magadan Regional Hospital was purchasing a CAT scanner through the assistance of the Institute for Circumpolar Health Studies.

Their refurbished General Electric CT 8800 had just been installed prior to my first visit in March 1990. At that time, it was the only CAT scanner in the entire Soviet Far East. It was not operational yet, but I spent my time learning and teaching as much as I could in their radiology department. I had brought over several donated radiology textbooks and several cases of iodinated contrast.

During my second visit in July, 1991 I settled into a daily routine, spending three to four hours in the CT department while patients were being scanned. Dr. Alexander Korovin and I sat together at the console, reading the images using a combination of Russian, English, Latin and "pointing-fingers-at-the-pathology". We learned a lot from each other and established, I believe, a professional and personal friendship.

It is a real credit to the Russians, especially their CT engineer, Gennady Khokhorin, that they keep this high-tech piece of equipment functioning day after day without readily available service or spare parts. Just about everything is in short supply. X-ray film is hand-dipped because they have no automatic processors. The X-ray film itself is in such short supply that only selected images from a CT study are photographed, the rest of the study being interpreted from the image on the console and the non-photographed images being stored on tape.

I was fortunate to be able to reciprocate during November and December of 1992 when Dr. Alexander Puzin, the senior CT Radiologist at the Magadan Regional Hospital, made his first visit to America, spending two weeks in Anchorage studying CT imaging, along with some MRI imaging. He spent several days at the Alaska Native Medical Center watching how CT procedures were done at our institution. Afterwards, we would read the cases together. He spent a day each at Humana, Elmendorf, Providence and the Anchorage Diagnostic Imaging Center. He gave a one hour conference at Humana showing interesting CT cases from Magadan and talking about radiology in the Russian Far East. It was a very busy and productive two weeks.

I was pleased and honored to sign an agreement between the Radiology Departments of the Alaska Native Medical Center and the Magadan Regional Hospital to continue our mutual cooperation. The aims of our agreement (translated from the Russian) are as follows:

1. To learn about modern methods of radiation diagnostics, including computer tomography.
2. To exchange experience in radiation diagnostics.
3. To study diagnostic work at a number of hospitals in Anchorage.
4. To establish in the near future, by way of modern communication facilities, permanent contacts and exchange of consultations on the interpretation of X-ray photographs.



Dr. Innokenti Yerilov reviewing x-ray at Magadan Regional Hospital

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# Proceedings of the Alaskan and Russian Natives' Health and Social Issues Conference Alaska

David L. Marshall<sup>(1)</sup>

Susan Soule<sup>(2)</sup>

## ABSTRACT AND SUMMARY

An Alaska-Russia Native People's Health and Social Issues Conference, sponsored by the Alaska Department of Health and Social Services, the Alaska Native Foundation, the University of Anchorage Institute for Circumpolar Health Studies, The International Scientific Center 'ARTIKA' (Magadan, Russia), the Associations of Native People of Chukotka and Kolyma, and the Magadan Native Association, was held in Wasilla, Alaska in May, 1992. The conference brought together Native people, primarily health and social services workers, to discuss differences and similarities in issues and approaches, and to lay the foundation for future collaboration.

The primary participants came mostly from rural villages and small regional cities, and represented Native Health Corporations, Native Associations, and villages. Additional participants came from the University of Alaska, the Alaska Department of Health and Social Services, the Indian Health Service, the Magadan Health Department, the Inuit Circumpolar Conference, and the International Circumpolar Health Union. A total of 39 people participated, including: eight Russian Natives (Chukchi, Even, and Siberian Yup'ik); three non-Native Russians; 18 Alaska Natives (Aleut, Athabaskan, Inupiat, Siberian Yup'ik, Yup'ik); nine non-Native Alaskans; one Canadian.

The issues discussed in individual and panel presentations, and in small groups, included history, demography, settlement patterns, the cash and subsistence economies, mental and physical health (epidemiology, etiology, treatment and prevention), education, governance, culture and language. As the conference participants came to know each other better, the discussions became increasingly open, and,

particularly around shared feelings of cultural oppression and loss, emotional.

Participants agreed that the Native cultures have more in common with each other than they do with either Russian or American non-Native cultures, that they have shared similar losses and face similar problems. They closed the conference by composing a joint statement the theme of which was the cultural foundations and inter-relatedness of social, health, and political problems, and the need for local, Native, control, over policies, land, and education, health, and social services programs.

This paper, organized by topic, summarizes the conference presentations and discussions, drawing on supplementary written materials for additional data to offer more detailed information in some areas.

## SETTLEMENT

Natives of the Russian Far East in the Magadan Oblast consist of Chukchis, Eskimos (Siberian Yup'ik), Even, Koryak, Yukhagir, and a few smaller minorities. The Chukchi predominate.

In the New World there are believed to have been three main waves of settlement after migrations across the Bering Land Bridge 11-15,000 years ago: the Indians, who spread out to North and South America; the Aleut, Inuit, and Yup'ik, who settled Alaska and Canada; the Athabaskans and the Tlingit-Haida, who occupied Interior and Southeast Alaska.

For thousands of years these indigenous inhabitants evolved cultures as central-based wanderers, based on the subsistence use of natural resources. In Russia, reindeer were central to the Chukchi for millennia, and are still the basis for their nomadic reindeer herders. In Alaska, reindeer were unknown until the late 19th century, and have always had a limited role.

Change occurred with the arrival of non-Natives: White Russians into the Magadan Oblast; Russians, Americans, and British into Alaska in the 19th and 20th centuries

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especially. They included fur traders, whalers, missionaries, miners, and political prisoners, who brought cash, goods, diseases, and different customs and attitudes. Change in Alaska occurred relatively quickly after the territory was sold by Russia to the United States in 1867.

## AREA, POPULATION, AND RESOURCES

Alaska and the Magadan Oblast are sparsely populated. They are about the same size (1.5 and 1.2 million km<sup>2</sup> respectively), and have about the same population (550,000 and 540,000 respectively). Non-Natives predominate in both areas. Alaska has 86,000 Natives, who are 16 percent of the total population; the Magadan Oblast has 21,000 Natives, who form four percent of the population.

In both areas the bulk of the Natives live in small communities. Most of Alaska's Natives-50,000 of the 86,000, or 60 percent-live in 176 small communities (166 with fewer than 1,000 people). There, they predominate: in most of the 176 small communities they account for 75 percent or more of the population.

Corresponding numbers were not available for the Russian Far East. Alaska's population rose by 37 percent in the last decade: from 400,000 to 550,000. Native population rose slightly less, by 34 percent: from 64,000 in 1980 to 87,000 in 1990. The state's non-Native population fluctuates around an upward trend: people immigrate from the continental U.S. in response to job opportunities, and emigrate when the economy and jobs decline. Even though there is population growth among Natives in rural and urban Alaska, Natives are migrating from rural to urban Alaska: rural Natives accounted for 40 percent of the state's Native population in 1980, and for 35 percent in 1990.

Corresponding population numbers were not available for the Russian Far East. The death rate among the indigenous peoples of the Russian Far North (which includes the Russian Far East) increased in the 60s, leveled off in the 70s, and appears to have dropped in the 80s. Within this overall trend, the death rate has increased among those aged 25-50 so that by the late 70s the death rate among them was four to six times as high as in the Russian population taken as a whole-indeed, it was "... higher for [them] than it was ... at the end of the last century." And, their birth rate has fluctuated. It declined

in the 60s, leveled off then rose in the 70s, and has declined from the mid-80s. The pattern among non-Natives is similar to their pattern in Alaska. Their numbers have increased (probably with less fluctuation in the Russian Far East). Now, with growing inflation which cuts into the Northern cost-of-living allowance, and with increasing economic uncertainty, numbers of non-Natives have begun to leave the settlements of the Russian Far East for western Russia where they have relatives, friends, and property. This movement is similar to the exodus of non-Natives from the Anchorage area especially, when the state's economic boom collapsed in the mid-80s.

Both areas export natural resources. Alaska exports minerals (oil and gas, gold, silver, lead-zinc, and some coal), fish, timber, and the tourism experience; oil and gas predominates. The Magadan Oblast exports minerals (gold and precious stones), reindeer products, and some fish; gold predominates; it does not have timber or a tourism industry.



Alaska Native Puppet show on AIDS and health

## TRANSPORTATION

The major transport modes are water and air. Marine transport-barges, freighters, and icebreakers in Russia's easternmost ports-is the major mode in both areas for exporting natural resources and importing fuel and heavy freight: air transport is used to carry almost all food and light freight into, and passengers into and out of, rural areas. Planes are used in Alaska, where all communities-even

those with fewer than 100 people-have airstrips; huge helicopters are used throughout the Magadan Oblast, where there are airstrips in only the larger communities. Alaska has a railroad connecting Seward on the south and Fairbanks on the north, through Anchorage, which is the biggest city, with 40 percent of the state's population. The Magadan Oblast has no railroad. Magadan, with 175,000 people, is its biggest city. Alaska has some highway mileage, which connects with the continental U.S.; the Oblast has some highway mileage in and around the City of Magadan. Alaska has a state-run ferry system serving the communities of Southeast and Prince William Sound.

## POLITICAL STRUCTURE

Alaska has 15 boroughs that cover almost half the state's area and that include the seven major urban areas.

But three of the 15 (Bristol Bay, North Slope, Northwest Arctic) consist mainly of rural villages. There are 150 cities with different powers to tax and spend, and about the same number of unincorporated communities. Most of the 150 cities and the unincorporated communities are small Native villages, which have two councils-municipal councils and traditional councils. The municipal councils, to which non-Natives can be and are elected, are eligible for some kinds of grants and loans from the state and federal government; the traditional councils, to which only Natives can be elected, are eligible for other kinds of grants and loans from the state and federal government. In small villages especially some Alaska Natives sit on both councils. There are 13 Native for-profit regional corporations started in the early 70s with money and land from the state and federal governments. All Alaska Natives are shareholders in one of the 13, which cover the whole state. Mostly, they invest in property and businesses outside rural areas. Some make a profit and some don't. All Natives who sit in the state legislature in Juneau, the state capital, were elected by voters from rural areas where Natives predominate. A few legislators from rural areas are non-Native. The state's two Senators and one Congressman in Washington DC., the national capital, are non-Natives.

The Alaska Federation of Natives is a non-profit organization that works to help resolve Native issues at federal, state, and local level. Another Native non-profit was formed recently around the theme of sovereignty; it takes part in international conferences on issues involving the world's people of the north, and presses for state and federal recognition of Alaska's Natives as sovereign tribes.

The Russian Far East has councils or executive committees at the level of villages, cities, regions, and oblasts. At the Oblast level the 100-member Magadan Regional Council-the counterpart of the Alaska state legislature-was established in 1990. The 100 members represent 100 regional election districts roughly proportional to the distribution of the population; for example, the City of Magadan had 30 districts. Natives can be and are elected at all these levels, but are always in a minority because they are a minority of the population at all these levels. They have Native representatives also at the Congress of People's Deputies-the legislature for Russia as a whole. In the last few years Natives have formed organizations to help represent their interests and to act

as a forum where they discuss common concerns. The Association of the Little People of the Magadan/Kolyma is the organization for Natives of the Russian Far East at the level of the oblast. The Magadan Native Association, and others, represent more-local groupings.

Until the Russian Communist Party was dissolved in 1991 there were two type of organizations which made economic, social, and political decisions: the executive committees; the party committees.

Four international groups provide a forum for the discussion, refinement, and representation of issues important to Alaskan and Russian Natives: the United Nations Working Group on Indigenous Peoples; the International Circumpolar Conference; the International Labor Organization; the International Union of Circumpolar Health. They are dealing with issues of sovereignty, empowerment, language, health, and others.

## THE CASH AND SUBSISTENCE ECONOMIES



Native delegation from Chukotka

The cash economy is well-developed in Alaska and in the Oblast. Cash rather than barter is the medium of exchange. It enters the areas' economies in exchange for the export of resources, as wages, and as transfer payments (money given to individual people by the government). Cash circulates within the areas as the residents buy goods and services from each other, and leaks out

as they buy goods and services from other parts of the country or abroad. Both Alaska and the Oblast have an extensive system of subsidies. Housing subsidies are common to both areas. Many subsidies are available in one area but not in the other, reflecting the fundamentally different economic and political structures of the two countries.

By definition, the government is the major economic actor in the socialist economy of the Russian Far East; it is a major player also in Alaska's economy, and is the major player in rural Alaska. The state is a major player in Alaska because of its oil-royalty revenues especially: they account for three-quarters or more of its revenues. In both areas, there is an extensive system of government payments to support rural inhabitants: wages to government workers; subsidies; pensions; cost-of-living adjustments; welfare payments; other transfers, such as dividends, in Alaska especially. Data on per capita incomes



as a measure of the standard of living were not presented or compared: the Oblast has an extensive program of subsidies-food and rent, for example-that makes such comparisons difficult.

The distinction between the cash and subsistence sectors is clear in Alaska but not in the Oblast, mainly because of differences between the two areas in the ownership of the things needed to harvest subsistence resources.

In Alaska the residents own their own gear and equipment: fish camps; boats; outboard engines; nets; snow machines; all-terrain vehicles; guns and ammunition. With the purchase of a license for hunting, fishing, and trapping they are free to do so subject only to state and federal restrictions on the amount of harvest (bag limits), on when they can do so (hunting periods), and in some cases on equipment (maximum outboard engine size, for example). The harvest is for personal consumption: it may not be sold commercially. Numerous studies have estimated that the subsistence harvest accounts for half or more of the food (protein intake) in the villages of rural Alaska.

In the Oblast the gear and equipment is owned not by individuals but by the collective farms which harvest reindeer, fish, and other marine resources, primary and processed, for local consumption and for export to domestic and foreign markets. No data are available on how much of the protein intake in the settlements of the Russian Far East comes from the collectives, although it is believed to be high in villages with Natives, since they are the reindeer herders and marine mammal hunters. One observer commented that in his region-Chukotka '... we've been hunting more freely for three years ...'

## EMPLOYMENT

In the Oblast, most of the rural jobs are on the collective farms: reindeer, sea mammals, crops, and supporting activities; other major sectors of economic activity include administration (broadly defined to include education and health as well as the work of political councils and their staff), construction, and transportation. For Natives of the Russian Far East, collectivization of agricultural activities and in some cases forced relocation changed their way of life in profound ways cultural and social. Herds of reindeer individually-owned and

raised for local consumption-a method of organization central to the way of life of the Chukotka and Kamchatka nomads-were organized into collectives with the animals raised for sale. Now, the long-run prospects for reindeer herding are unclear, because fewer Native women are willing to accept the nomadic living conditions. Gold mining, which is widely dispersed in rural areas, has '... sometimes ... led to tragedies [for] traditional, Native settlements in the very heart of these gold mining [areas].' The tragedies are from cultural and social disruption caused by the influx of non-Natives, alcohol, cash, different jobs, and pollution of the environment.

In Alaska, most of the steady rural jobs are also with the government: in education, health, administration, law enforcement. Most of these jobs are held by non-Natives even in villages that are predominantly Native because non-Natives have higher levels of formal education. Native non-profit corporations also provide numerous steady jobs based on government grants. Most of the non-government rural jobs are seasonal rather than steady, being based on commercial fishing especially, and also

on timber in Southeast. The North Slope Borough is unique in having its own source of funds-taxes on oil-production property in Prudhoe Bay. The state government supports some summer construction jobs. Alaska has a relatively small agricultural crops and reindeer sector. Oil and gas production is concentrated, not dispersed, and has had no adverse effects on the Native population. On the North Slope, where the bulk of the oil is produced,

it is pumped from underground to the across-state pipeline with little damage to the local environment. The production complex is a self-contained enclave supplied by air from Anchorage and Fairbanks, and has no links with the Native villages on the North Slope except that the few Natives who work there fly back and forth from home to work. One Alaska Native said that

Hard information on rural underemployment and unemployment is not readily available. The problem is known to be extensive in rural Alaska. The Alaska Department of Labor routinely publishes numbers estimating unemployment at 15 percent, but notes that this is an underestimate because it doesn't include people who are out of work and who have stopped looking for work locally. One Native Alaskan said that 'The majority of the villages have really high unemployment [especially in fall, winter, and spring].' One researcher estimates it



Alaska/Russia Natives helping each other make it through the day



at 50 percent and up. No numbers are available for the settlements of the Oblast. One resident said '... We can't say that we do have unemployment ...' but pointed out that 'A lot of young people are just simply idling. ...' The better-educated, who are generally non-Natives, get the steady jobs. But there is work-even if unskilled-for younger workers, and/or for the less well-educated who tend to be Natives. One Russian Native noted that her brother was unable to find work fitting his training as a skilled mechanic, and so went into reindeer herding. Another said that the ones idling are returnees from schools, which '... didn't teach our people to work hard. ...' One Russian scholar (not at the conference) who specializes in the indigenous peoples of the Russian North states that 'Unemployment has become a [permanent] social phenomenon in the indigenous villages.'

### EDUCATION

Both areas have kindergarten, grade school, and high school. In Russia, streaming occurs at age 11 or 12: students who pass the exams go on to higher education intended to lead to university; those who fail are enrolled in vocational education institutions. In Alaska there is no such streaming. In Alaska, all villages-even those with fewer than 100 people- have grade-school and high-school education. In the Oblast, high-school education is not available in the villages; rural students are enrolled in urban schools.

In the villages of rural Alaska and the Russian Far East most teachers are non-Native even though in rural Alaska most students and school board members are Natives; in the settlements of the Oblast, both rural and urban, Natives are in the minority.

In the Russian Far East most Natives have eight years of schooling, some have 11, and a few-the gifted-go to college. But half drop out of college early, and few graduate. Corresponding numbers were not given for Alaska's Natives, but one pointed out that there is a different pattern among Natives depending on their sex: most young Native women-80-90 percent-at least enroll in institutions of higher education after high school, whereas half the young men stay home. But in Alaska too, many more Natives drop out than graduate from college.

One Russian Native observer said separation from her children-she living and working in a rural area, they in urban schools-is '... very painful ...' but typical for rural parents in the Russian Far East, and noted that in their schools the students '... get into drinking and smoking and all those bad habits ...'

One Alaskan observer reported on conversations with Alaska Natives who said that before the villages had their own high schools they went to boarding schools for extended periods, and so missed the time of life when they would have learned parenting skills from their parents.

## HOUSING

Housing in the Russian Far East, as elsewhere in Russia, consists mostly of concrete-block apartments. In the smaller rural settlements single-family wooden houses and cabins are found. In general, Native housing is inadequate in quantity and quality, without running water or toilets. The 464 reindeer herders who tend the 45 herds in the Olskiy Region east of Magadan have no permanent housing except the yarangas of the tundra. As a result they have high rates of mortality from TB. In the villages of the Chukotka region it is not uncommon for three generations to live together overcrowded in one small housing unit. This contributes to the high death rates among children and young people. Again, one observer said she has had to live with her parents for 11 years because she cannot get an apartment in her village.

In Alaska's rural villages small single-family wooden houses are the norm, some built with local materials inland where there is timber, most in recent years built by the U.S. Department of Housing and Urban Development using prefabricated materials including plywood and sheetrock.

In the Russian Far East housing is subsidized for Natives and non-Natives. In Alaska many Natives live in housing subsidized because they fall below poverty levels, and the many non-Natives who are teachers in the villages are subsidized by the state Department of Education through their school districts. Continuing building in rural Alaska of new housing units, and rehabilitation of the old, indicate that the stock of housing was inadequate in quantity and quality. One observer characterized village housing as cold, and normally without running water or flush toilets, which are installed only in the school buildings and the apartments where the teachers live.

## MORTALITY, HEALTH, AND ALCOHOL

In both Alaska and the Russian Far East, Natives were decimated by diseases introduced by non-Native carriers. In the Oblast, this has continued as Native parents return to the villages from urban areas where they have visited their children in school, and as the shift from traditional to modern clothing has increased the incidence of pneumonia.

Among Natives of the Russian Far East, alcohol-related accidental deaths are the leading manner-of-death; cancer is in second place; third is alcohol poisoning; suicide is seventh. Among Alaska Natives aged 10-40 years, accidental deaths, suicides, and homicides were the three leading manners-of-death in that order in the 80s, among males especially; heart diseases and neoplasms predominate in those aged 40 and up at time of death. One Alaska Native speaker said that

'... the young men seem lost, threatened by cities, we lose them right after high school...'

Natives have a markedly lower life expectancy than non-Natives in the Magadan Oblast: 43 years for Native males and 47 years for Native females, compared with 58 years for all males and 68 years for all females. In Alaska the difference is much less: 68 for Natives and 73 for non-Natives. And, it has risen in Alaska, for Natives especially, since 1950, when life expectancy was 48 for Natives and 67 for non-Natives.

The improvement in Alaska is from an improved standard of living, and from better health care which has reduced especially deaths from infant mortality and communicable diseases. But deaths from alcohol-related accidents and suicides—particularly among young Native men—have risen dramatically, so that the Native death rate actually increased slightly over the period 1970 to 1980. Similarly among the peoples of the Russian North: deaths from accidents, suicides, and murders replaced deaths from disease between 1950 and 1980. In short, mortality among Native people in Alaska and in Russia now reflects social conditions rather than improvements in medicine. One consequence is an increase in the number of single-parent (mother only) households which, it was suggested, by one participant, perpetuates the problem for young men without a father figure and raises the chicken and egg question: if a young man is handicapped without a father but the mother doesn't want the father as a partner because he drinks, and/or is abusive, where is the cycle to be broken?

It was suggested by one Russian Native speaker that the causes of alcohol abuse and alcoholism include separation from families (reindeer herders), lack of work, lack of hobbies—not knowing how to spend free time. Another, describing her people, said that the Siberian Yup'ik males were hunters but that now, since hunting is secondary, they have lost their roots so that now many '... big healthy men are parasites and the women take care of them.' Yet another explained the difficulties created by political leaders and businessmen who take alcohol to the reindeer camps, which are normally dry, and by reindeer herders who drink to excess when they leave their camps for short visits to their villages. One Alaska Native speaker also notes a link between mental health, traditional activities such as subsistence harvesting, and work: 'One of our biggest problems has been that the government in America has not recognized subsistence hunting [and] fishing as a job, and that's caused some of the mental health problems that the men have had.'

## HEALTH CARE

In the Russian Far East the main hospital is in Magadan, and there are smaller hospitals in both urban and rural settlements even for very small communities. Typically,

the small hospitals have a doctor and one or two assistants, and half a dozen beds. Helicopters can fly in and out on a moment's notice for emergencies. Health care for the nomadic reindeer herders is also nomadic: a mobile team including a doctor, a dentist, and assistants, visits the camp sites in summer, takes x-rays, deals with injuries, advises on diet. (Since summer is short in the High Arctic, the mobile teams often set up their camps before the spring snow melts, and are there after the first snow falls at the end of summer.)

In Alaska the major urban areas and regional nodes (Bethel, for example) have hospitals including one (in Anchorage) for Native patients only. The villages have clinics staffed by health aides. Emergency plane transport is available on call. The federal government pays the cost of health care for Natives. Non-Natives rely on health insurance—monthly premiums paid to the insuring company which then pays the medical bills. There are 13 Native regional health corporations well-staffed with Native and non-Native specialists in physical and mental health, funded by an extensive network of state and federal grant programs including especially those of the Alaska Department of Health and Social Services and the U.S. Indian Health Service.

## ISSUES

As one Alaska Native spokesman put it, 'Most of Alaska is worried about money—the shortage of money. The Native communities are modern enough where they are worried about the shortage of money too. We do adapt to western values. But we are also concerned about health. We are concerned about land. We are concerned about natural resources. These are [our] hunting and fishing resources. We are worried about education and almost everything else you can think of.' These same themes loomed large in the Russians' presentations.

As regards money, the threat in Alaska is from reduced revenues as production of oil from the Prudhoe Bay supergiant deposit declines over the next decade to level off at a small fraction of its present annual output. This is the source of state revenues, and state spending is the source of money in rural Alaska. In the Russian Far East, the shift from a command to a market economy has brought inflation without an increase in production. This is increasingly creating hardships in rural Native communities whose residents had little surplus and limited incomes to begin with. Russia's system of state-subsidies for housing, food, and other things has in essence collapsed.

As regards health, the increase in alcohol-related accidental deaths and suicides among the young Native men in both Alaska and the Russian Far East is seen as a symptom of a profound and puzzling malaise in the face of living and medical care standards that in recent



decades have risen in Alaska and that may have risen in the Russian Far East. Mortality is now a social rather than a medical problem. Both groups relate this to the loss of cultural and social traditions, and language, with the arrival of non-Natives who have a dynamic, individualistic, future- and cash-oriented society (infused early with missionary zeal, alcohol, and diseases), as compared with the static, subsistence, group- and present-oriented way of life typical of Native cultures.

As regards land, the destruction of land and waters from gold mining in the Russian Far East is seen as a deep-rooted problem for the development and maintenance of a prosperous agricultural, fishing, and reindeer sector even if privatization and its related incentives replaces collectivization. It is expected that there will be pressure to increase surface mining of minerals, and oil exploration, as western companies seek profitable opportunities abroad where environmental regulations are less well-developed. In Alaska the concern is over the protection of the subsistence resources vital to rural Natives given the unresolved conflict between federal law (which establishes rural preference over the harvesting of the resources) and state law (which makes discrimination in favor of any one group unconstitutional).

As regards education, separation of children from parents in the Russian Far East is seen as a major problem for parents who are distressed by the separation and for students who are exposed to unhealthy social forces in the urban areas where the influence of their parents is not available. In Alaska the concern is over whether or not rural Natives are getting the education they need to occupy steady jobs in their villages or in urban Alaska, and over the high rate of suicides among young Native males just out of high school who are not yet working.

The participants' ideas about programs to address the problems focused on both treatment and prevention. A medical model was rejected, however, in favor of models that addressed social, emotional, spiritual, and cultural issues, as well as the issues of physical health. While there was general agreement that rapid, and, to a greater or lesser degree, forced cultural change is a major contributor to social problems, to substance abuse, and to injury-related morbidity and mortality, Alaska Natives, to a far greater extent than Russian Natives, have developed numerous culturally-based treatment and prevention programs.

One Alaska Native described his region's 'Fish Camp Recovery Programs', where recovering substance abusers spend some weeks or months living in the traditional manner, practicing traditional skills, reconnecting with traditional values, rebuilding individual and cultural pride. Others described village and regional prevention programs which employ Elders to teach traditional values and skills, and which emphasize building pride and self-esteem through a rebirth of knowledge of the Native culture.

## CONCLUSION

Both groups believe that success in dealing with these issues will take time and cooperation among Natives and non-Natives. The Alaska Natives feel hopeful, because they know that they are now dealing more openly and successfully with alcohol, sexual abuse, and violence in their villages, in part because of a shift in the kind of help offered by state agencies such as the Department of Health and Social Services. The shift is towards greater local control over the kind of programs, both preventive and treatment, that deal with these symptoms of malaise. They hope that increasing self-awareness and control will over time be translated into greater control over the kind and pace of influences from outside their communities that do harm in them. The Natives of the Russian Far East think they have a longer and a deeper row to hoe because of the great damage done to their way of life during and since the Stalin era, and because of the uncertainty and turmoil created by the economic, political, and social revolutions stemming from and glasnost. They hope to cooperate with their Alaska cousins, who seem to be further along in social and political independence, in the years ahead. Both groups expressed great pleasure at having come together for a week-long conference, were struck by the similarity of the issues they face, and realized that their Nativeness created a bond that overcame differences of language and nationality.

The participants concluded the conference by sharing perceptions of each other, and drafted a joint statement.

The perceptions reflected the warmth and good feelings which developed over the five days. Russian Natives expressed surprise that Alaska Natives were not, as they had thought, 'reserved', but, rather, 'smiling, good-natured, and very relaxed.' Alaska Natives noted that the Russians were 'initially reserved, but once they get the opportunity to be relaxed and share, their stories and ideas flow endlessly, [and] they have a wonderful commitment to family, community, culture, and country.'

The joint statement reflected common concerns regarding health and social issues, ownership and control of land, economic development, education, political rights, and preservation of Native cultures. 'The Native people of Alaska and the Russian North are determined to live proudly as Native people, retaining traditional cultural values, language, and ways of life [and] control of our own destinies in all areas.' The statement asked all levels of Russian, American, and Alaska government to recognize 'Native tribes, villages, and associations as governmental and political entities, [with] power to enact and enforce laws and regulations at the community and tribal level, [with] adequate financial support to carry out locally determined policies and programs, [with] permanent ownership and the right to make final determination of the use of all natural resources of their ancestral lands.'



The statement further noted that 'The Native people of Alaska and the Russian North are the same people, but have been separated by artificial and arbitrary political barriers', and concluded with a strong request for moral and financial support for 'efforts by the Native people of the Russian North and Alaska to work together to address their common problems.'



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# Self-reported Illness Among Travelers to the Russian Far East

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## SYNOPSIS

This study evaluated the risk of travelers to the Russian Far East developing acute gastrointestinal or respiratory symptoms. Passengers and crew on 10 commercial airline flights from the Russian Far East to the United States were asked to complete a health questionnaire that asked age, sex, country of residence, length of stay, foods and beverages consumed, and about gastrointestinal or "flu" symptoms. Questionnaires were returned by 353 of 662 persons (53.3 percent).

The most frequently reported symptoms were diarrhea (N = 18; 5.1 percent) and "flu" symptoms (N = 15; 4.2 percent). Among those people who reported symptoms, most were sick for 3 days or less, although 10 (27.0 percent) were still sick at the time that they entered the United States. Age and sex were not associated with symptoms. Persons who drank untreated tap water were more likely to have gastrointestinal symptoms (relative risk = 2.7; 95 percent confidence interval = 1.2, 5.9) while those who drank bottled or canned fruit juice were protected (relative risk = 0.4; 95 percent confidence interval = 0.2, 0.8).

The incidence of "flu" symptoms was similar to the rate for the general population of the United States while the incidence of gastrointestinal symptoms was increased and only slightly less than the rate among travelers to developing countries. Travelers may wish to restrict consumption of untreated tap water and increase consumption of fruit juices.

Additional work is needed to identify the pathogens responsible for acute illnesses among travelers to the Russian Far East.

The recent historic changes that took place in the former Soviet Union led to the initiation of regularly scheduled commercial air travel between the Russian Far

East and the United States in 1991. The relaxation of travel restrictions in the former Soviet Union and the emergence of business opportunities in the region have resulted in increasing numbers of visits by American citizens and other foreigners. We are not aware of any studies examining the potential risks of communicable disease transmission to such travelers.

In June 1991, we investigated two apparently unrelated cases of salmonellosis in U.S. citizens who had recently returned from the Russian Far East city of Khabarovsk. Although no specific source for these infections could be identified, this occurrence raised concern that other persons traveling to Russia might be at risk of contracting infectious diseases.

To evaluate better the health experiences of Americans traveling to the Russian Far East, we conducted a survey of persons arriving by air in Anchorage, AK, from Russia. The survey was designed to assess rapidly the presence of and possible risk factors for acute gastrointestinal and upper respiratory symptoms.

## METHODS

We made arrangements with a commercial air carrier (Alaska Airlines) for airline personnel to distribute a health questionnaire to all passengers and crew on each flight arriving in Anchorage from Khabarovsk and Magadan in the Russian Far East between June 12 and July 8, 1992. Due to an oversight, questionnaires were not distributed on 2 of the 12 flights during this interval. The anonymous, self-administered questionnaire included questions on the passenger's age, sex, usual place of residence, types of foods and beverages consumed in the Russian Far East, and length of stay. Each person was asked to indicate which, if any, of the following symptoms developed while in the Russian Far East:

- (1) Medical Epidemiologist, Section of Epidemiology, Division of Public Health, Alaska Department of Health and Social Services.
- (2) Nurse Epidemiologist, Section of Epidemiology, Division of Public Health, Alaska Department of Health and Social Services.

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- nausea or vomiting (lasting at least 12 hours),
- diarrhea (three or more loose or watery stools in 24 hours),
- abdominal pain or discomfort (lasting at least 24 hours),
- fever or "feverishness" (lasting at least 24 hours),
- "flu" symptoms - fever, headache, sore throat, body ache (lasting at least 24 hours).

For those persons having at least one of these symptoms, the questionnaire asked how long after arriving in the Russian Far East the first symptom developed, whether or not the symptom(s) lasted more than 3 days, and if the person was still sick. All questions (except age) were answered by checking forced-choice boxes to indicate the applicable response. Completed questionnaires were collected at the Anchorage International Airport.

A wallet-size tear-off card was attached to each questionnaire for the traveler to keep. The card requested the person to telephone (collect) the Alaska Division of Public Health if any of the diarrhea, abdominal pain or discomfort, or fever or "feverishness" symptoms previously described developed within 15 days of return to the United States. Statistical evaluation of completed questionnaires was conducted with Epi Info computer software (1).

## RESULTS

Questionnaires were returned by 353 of 588 passengers and 74 crew members for an overall response rate of 53.3 percent. Respondents were significantly older (mean age = 52 years) than the U.S. general population and were more likely to be male than female (table 1). Of 351 people who indicated their usual place of residence, 344 (98.0 percent) were from the United States and 7 (2.0 percent) were from other countries; none were from the former Soviet Union. Of 352 people who indicated the

Table 1. Demographic characteristics of 353 travelers arriving by air in Anchorage, AK from the Russian Far East, June-July 1992

Category	Number	Percent
Sex:		
Male	203	58
Female	149	42
Not specified	1	0
Age (years):		
Younger than 30	18	5
30-39	59	17
40-49	90	25
50-59	51	14
60-69	71	20
70 and older	61	17
Not specified	3	1
Place of residence:		
Alaska	53	15
United States (not Alaska)	291	82
Other country (not former Soviet Union)	7	2
Not specified	2	1

length of time they spent in the Russian Far East, 305 (86.6 percent) were there for 14 days or less.

Symptoms were reported by 37 (10.5 percent) of the returning persons; diarrhea (N=18) and "flu" symptoms (N=15) were most frequently reported (table 2). Eight travelers reported having more than one symptom; six persons had two symptoms, and two persons had four

Table 2. Symptoms of 37 travelers returning from the Russian Far East who reported having symptoms, June-July 1992

Symptom	Number	Percent
Diarrhea	18	49
"Flu" symptoms <sup>(1)</sup>	15	41
Nausea or vomiting	8	22
Abdominal pain or discomfort	5	14
Fever or feverishness	3	8

(1) Defined as fever, headache, sore throat, or body ache lasting at least 24 hours.

symptoms. Only two people had both gastrointestinal (nausea or vomiting; diarrhea; or abdominal pain or discomfort) and "flu" symptoms.

Although the proportion of travelers who reported having symptoms was greater among those younger than age 50 (23 of 167; 13.8 percent) than among older people (14 of 183; 7.6 percent), this difference was not statistically significant (relative risk [RR] = 1.8; 95 percent confidence interval [CI] = 1.0, 3.4). Likewise, there was no association between sex and symptoms (RR for male versus female = 1.0; 95 percent CI = 0.6, 1.9). Persons who reported that they lived in Alaska had a greater risk of becoming ill than those residing in other States (10 of 53 Alaska residents indicated that they had symptoms versus 27 of 291 persons from other States, RR = 2.0; 95 percent CI = 1.1, 4.0).

Of persons who reported having one or more symptoms, 24 (64.9 percent) indicated that symptoms lasted 3 days or less, 9 (24.3 percent) indicated they lasted more than 3 days, and 4 (10.8 percent) did not specify how long symptoms lasted. Ten of the 37 ill persons (27.0 percent) indicated they were still ill at the time they completed the questionnaire. Twenty persons (54.1 percent) who became ill had onset within 2-6 days after arriving in the Russian Far East. Nine (24.3 percent) became ill earlier and seven (18.9 percent) later than this.

There was no relationship between the length of stay and gastrointestinal symptoms: 7 percent of persons who spent either less than 7 days (11 of 152) or 7 days or more (13 of 200) in the Russian Far East reported gastrointestinal symptoms (P = .79) (table 3). The proportion of travelers reporting "flu" symptoms, however, increased



Table 3. Number of travelers reporting symptoms, by length of stay in the Russian Far East, June-July 1992

Days spent in Russian Far East	Number of travelers	Gastrointestinal symptoms <sup>(1)</sup>		"Flu" symptoms <sup>(2)</sup>		Any symptom	
		Number	Percent	Number	Percent	Number	Percent
Less than 1	11	0	0	0	0	0	0
1-2	20	5	25.0	0	0	5	25.0
3-6	121	6	5.0	3	2.5	8	6.6
7-14	153	8	5.2	6	3.9	13	8.5
More than 14	47	5	10.6	6	12.8	11	23.4
Not specified	1	0	0	0	0	0	0
Totals	353	24	6.8	15	4.2	37	10.5

(1) Includes nausea or vomiting, diarrhea, or abdominal pain or discomfort.

(2) Defined as fever, headache, sore throat, or body ache lasting at least 24 hours.

as length of stay increased (table 3). When the association between risk of developing symptoms and place of residence (Alaska residents had a twofold higher risk) was adjusted by controlling for length of stay, the point-estimate for the effect was decreased, and the 95 percent confidence interval included 1.0 (Mantel-Haenszel RR = 1.6; 95 percent CI = 0.7, 3.4).

There were associations between several of the types of beverages and food sources we asked about and gastrointestinal symptoms. Persons who reported drinking untreated tap water or who brought their own food to Russia had nearly a threefold higher risk of illness compared with those who did not (table 4). Those drinking bottled or canned fruit juice had an illness rate 2.5 times lower than that of persons who did not (table 4).

There was no association between any of the food sources we asked about and "flu" symptoms (table 4). However, persons who ate food that they purchased from Russian markets or shops (RR = 2.5; 95 percent CI = 0.9, 6.6) or who brought their own food (RR = 2.4; 95 percent CI = 0.9, 6.6) both had elevated relative risk estimates approaching statistical significance. These sources were examined be-

cause each may have served as a surrogate measure of the degree of socializing with Russian people.

Gastrointestinal symptoms were reported by 24 people (6.8 percent). Among 305 travelers who spent 14 days or less in the Russian Far East, 19 (6.2 percent) developed gastrointestinal symptoms. To estimate person-days of exposure, we assumed that each person was in the Russian Far East for the number of days equal to the mid-period of the indicated time interval. For example, the 121 people who stayed for 3-6 days (table 3) were

Table 4. Relative risks for gastrointestinal symptoms among 353 travelers associated with selected exposures, Russian Far East, June-July 1992

Exposure	Number exposed	Number ill <sup>(1)</sup>	Relative risk	95 percent confidence interval
Consumption of:				
Untreated tap water	56	8	2.7	1.2 - 5.9
Untreated surface water	22	3	2.2	0.7 - 6.7
Boiled or filtered surface water	27	3	1.7	0.6 - 5.4
Bottled water	291	20	1.1	0.4 - 3.0
Boiled or filtered tap water	238	16	1.0	0.4 - 2.2
Bottled or canned fruit juice	250	11	0.4	0.2 - 0.8
Coffee or tea	318	19	0.4	0.2 - 1.1
Sources of food:				
Hotels or restaurants	341	23	0.8	0.1 - 5.5
Private residences	137	10	1.1	0.5 - 2.5
Russian markets or shops	93	9	1.7	0.8 - 3.7
Carried to Russia	136	15	2.7	1.2 - 5.9

(1) Number of travelers reporting nausea or vomiting, diarrhea, or abdominal pain or discomfort among those with the specified exposure. Total number with any gastrointestinal symptom = 24.

assumed to have 544.5 person-days of exposure (121 persons multiplied by 4.5 days). Persons in the Russian Far East for 14 or fewer days had a total of 2,186.5 person-days of exposure for an incidence rate for gastrointestinal symptoms of 0.87 per 100 person-days (19 divided by 2186.5 multiplied by 100).

The incidence rate for "flu" symptoms was estimated in a similar way. Among those who spent 14 days or less in the Russian Far East, there were nine people who reported "flu" symptoms for an incidence rate of 0.42 per 100 person-days.

Eight passengers telephoned us to report onset of symptoms after returning to the United States. These people were not included in the 37 travelers who reported symptoms on the health questionnaires. Diarrhea was the most frequent complaint (N=6) followed by "flu" symptoms (N=2). Illness occurred from 0 (day of arrival in United States) to 8 days after returning; six of the eight persons reported onset during the first 3 days after return. Age ranged from 38-68 years (mean = 49 years); all were residents of States other than Alaska.

## DISCUSSION

This rapid health assessment indicated that 10 percent of short-term American travelers to the Russian Far East developed acute gastrointestinal or respiratory symptoms while in Russia. If persons having onset of gastrointestinal symptoms within 3 days of return to the United States were included, 12 percent of travelers had illness. During the time the survey was conducted, no cases of salmonellosis, shigellosis, giardiasis, or other infectious causes of gastroenteritis among persons who recently returned from Russia were reported to us. To our knowledge, this is the first study examining the incidence of gastrointestinal or respiratory symptoms among travelers to the Russian Far East.

The National Health Interview Survey (NHIS) estimated that the incidence of "indigestion, nausea, and vomiting" resulting in either medical care or activity restriction was 1.6 cases per year per 100 persons among noninstitutionalized adults ages 45 or older in the United States (2). Likewise, the Tecumseh study, a community-wide active surveillance program conducted in Michigan, reported that the incidence of enteric illness syndromes (vomiting, diarrhea, nausea, or upset stomach) was 51 per year per 100 persons among adults ages 50 or older (3).

Clearly there is a wide difference between these two rates, but if incidence was distributed uniformly over an entire year, the daily incidence rates would be 0.004 per 100 persons for NHIS and 0.14 per 100 persons for the Tecumseh study. These rates can be compared with the estimated incidence rate of gastrointestinal symptoms for travelers to the Russian Far East, 0.87 per 100 person-

days. The observed rate is slightly more than 6 times greater than that reported by the Tecumseh study and more than 200 times greater than NHIS.

Symptoms experienced by travelers seemed to have a relatively short course; nearly two-thirds of ill people reported that they were well within 3 days. Although the incidence of gastrointestinal illness was high when compared with rates in the United States, it is lower than rates reported in a recent review of diarrhea among travelers from industrialized countries to the developing world (4). The median incidence rates for diarrhea were 53 percent for travelers to Latin America (24 studies; median length of stay = 21 days) and 54 percent for travelers to either Asia (8 studies; median length of stay = 34 days) or Africa (3 studies; median length of stay = 28 days) (4). These rates convert to 1.6-2.5 per 100 person-days, only moderately higher than the rate of gastrointestinal symptoms we observed for travelers to the Russian Far East.

The estimated incidence of "flu" symptoms can also be compared with NHIS and Tecumseh study results. For persons ages 45 or older, NHIS reported incidence rates of 17.9 per 100 person-years for the common cold, 19.7 per 100 person-years for influenza, and 3.2 per 100 person-years for other acute upper respiratory infections (2). These rates correspond to 0.05 per 100 person-days for colds, 0.05 per 100 person-days for influenza, and 0.009 per 100 person-days for other upper respiratory conditions. Examination of data reported by the Tecumseh study results in a mean annual incidence for "total respiratory conditions" of 143 per 100 person-years for persons age 50 or older (5). This corresponds to a rate of 0.39 per 100 person-days.

Thus, the incidence experienced by travelers, 0.42 per 100 person-days, is higher than the rate reported by NHIS but similar to the rate reported by the Tecumseh study. NHIS included only illnesses resulting in either medical attention or restriction of usual activity, not counting persons with the mildest symptoms. Assuming that there were three to four people with respiratory symptoms for each person seeking medical care or limiting their usual activity as a result of these symptoms, the incidence of "flu" symptoms among travelers to Russia is probably similar to what would be expected in the United States based upon NHIS data.

In summary, Americans traveling to the Russian Far East appear to be at increased risk of developing gastrointestinal symptoms. The magnitude of this risk was slightly less than that reported among travelers to the developing world. The risk was highest among persons who consumed untreated tap water or brought their own food and lower among those who drank bottled or canned fruit juice. Although we could neither distinguish between various pathogens that cause such symptoms nor evaluate the efficacy of possible preventive measures, it may be prudent for future travelers to avoid consumption

of untreated tap water and to rely on bottled or canned fruit juice. Because the response rate was relatively low, the results might be different if passengers and crew members who did not return questionnaires differed from those who did. However, our methodology did not permit us to evaluate or follow up nonrespondents. The observed incidence of respiratory symptoms could have been higher if we had asked about "flu" symptoms that occurred after returning to the United States.

Further work is needed to confirm these findings, clarify the potential causes, and evaluate potential preventive measures. Currently, the number of American citizens traveling to the Russian Far East is small. If all seats on every commercial flight were occupied, fewer than 10,000 passengers per year would be transported between the west coast of the United States and the east coast of Russia. As relations between Russia and the United States continue to improve, the number of Americans visiting the Russian Far East is certain to grow, and it will become increasingly important to understand better both the health risk to American travelers and the potential for importation of infectious diseases into the United States.

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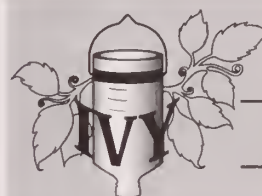
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# Social-political and Medical-psychological Aspects of Life of the Native People of the North

Larissa Abryutina, M.D. <sup>(1)</sup>

I live and work in Bilibino, Chukotka.

Chukchi, Even and Yukageer people are the indigenous inhabitants of this territory. Chukchi people traditionally were the reindeer herders, while the Even and Yukageer people have hunted, fished and herded reindeer as well.

The common aspect of their lives and labor was constant migration, which provided them with not only food, but the opportunity for contacts and consolidation of the isolated groups of nomads. When the Soviets time came, the lives of Natives changed seriously. The administrative division of autonomous regions, including Chukotka, was made. The regions were divided into districts. The pastures and land were divided between collective farms and state farms. The reindeer were collectivized in most cases against the owner's will and without refunds.

The indigenous people did not understand the reasons and the heart of the matter of this expropriation. Earlier they could have resisted, but now the forces were not equal. They were getting scared. They were afraid to even see the tracks of the "foreign people" - as the old men told me. Some of the nomads hid for a while.

For example, a person named Khabarovski hid with his herds in the Omolon tundra until 1970, when he was caught and sent to a psychiatric clinic. He was, I think, a wise and healthy man.

Native people were given new names and sometimes even a new nationality. They were excommunicated from their beliefs. The reindeer herders and their deer were divided into collective farms and state farms. At the same time, the families and communities of nomads were mixed together and turned into industrial brigades.

These brigades were assigned to pastures which were not very good. But what is very important, the pasture's boundaries were firmly drawn. As a result, nomadic migration was stopped, and the natural ways and technologies of herding and hunting broke down. The system of cooperation and connections of Native people with land and each other were broken as well. Ethnic occupations broke down. The Even and Yukageer people were forced into using Chukchi styles of reindeer herding. Newcomers (the Russian people) became hunters and fishermen, using all the traditional Even and Yukageer pastures.

Central farmsteads were founded for the coordination and supply needs of all collective and state farms. These were miniature base settlements, centered between a few reindeer brigades. Common sense says that if the aim of a new government was not to destroy but help people, those bases should have been centers of supply and helped develop the nomad's routes, along with the hunting and fishing industry, and found places where the Native people could live and work.

However, everything happened to the contrary. Only the settlements, some-

times poorly located, were developed. The "Russian speaking population" began moving to those so called Native villages. Administration and management, as well as service industry people, were basically these "Newcomers". With the increase of the population in the villages, the expenses for herd maintenance also increased. The number of reindeer herders is now only 10-20 percent of a village's population. The construction of medical, social and child care facilities, schools and boarding schools was done in the villages. At the same time, children were removed from their parents in the tundra. It was done hastily and insistently. The parents resisted, of course, but their resistance was overcome by force and deception.



Reindeer herder's camp on Bolshoi Anyui river, Chukotka

(1) Chairman, Native People's Association; People's Deputy, Chukotka Region; Chief of Mobile Medical Teams, Bilibino, Chukotka

For example, my younger brothers and I were moved to the village several times, and mom "stole" us and took us back to the tundra by deer. But all these efforts were done for nothing, and finally the plan to move the children from the tundra was completed.

Right after the moving of the children and women, the same procedure took place with the elderly people. Even now, most of the elderly do not have their own place to live in. Most men of an age able to work remained in the tundra.

The moving of children, women and old people from the tundra lead to the destruction of families and the traditional culture and style of life for a succession of generations. The tents made from reindeer skin (the traditional type of housing in the tundra), fur clothes and boots disappeared from everyday life. There was nothing done instead. The tundra turned into an odious place for men.

I think this was a consequence of the forced government policy and ideology intended "to abolish the archaic form of aboriginal style of life as inappropriate to the socialist style". The intent of this concept was to completely end the nomadic life, except where it was needed to support the new farm system. According to theory, the founding and development of the "central farmstead" in native villages had to play an important role in the stabilization and "the taking over of culture" of the former nomads. The Newcomers pushed this concept very hard through the farm's administration and management. But as a rule, these people were very far from the unique ethnic problems of the Natives. At the time of the creation of collective and state farms, alcohol was increasingly available, as well as the pressure to follow the New-comer's alcohol traditions. Even with some temporary letups, all these problems are still going on.

As a result, some practices of "the new culture", in trying to overcome "wildness" and change the native people over to a settled way of life, had many negative consequences. First, the limitation put on nomadic wandering, and in some cases even its prohibition, has hurt the firm and genetically fixed requirement to wander. Second, the system of traditional marriage patterns was broken, partially because the contacts between groups of nomads was ended, and because women were moving out from the villages. As a result, a lot of women turned out to be single parents or entered into mixed marriages.

Girls and young ladies became subjects of sexual abuse. This is, literally, the biological assimilation which nobody is talking about. Evidently, this is leading not only to the destruction of morality, but also to the mass destruction of genetic code. With Natives living in cities and villages, they have probably stepped beyond the possibility of their population having an ethnic rebirth.

There was also a requirement to have the medical people and managers of collective farms move pregnant women out of the tundra, to stay at the maternity ward one month prior to delivery and a month after. Maybe it reduced the mortality rate? But how was that reflected in the future of a child? Staying at the maternity ward could help coping with the bad living conditions and prevent some infections, but at the same time it influenced the immune system as well. The hospital environment is not normal for indigenous people, and always is a source of psychological discomfort, stress, a great change of nutrition, etc. Several times I hid "fugitives" from the maternity wards myself.

After a month of being in a hospital, the nomad's baby goes to the Native village. There, the baby's autonomic life is usually continued in a 24-hour child care institute, similar to the day care centers in the kindergartens and boarding schools. This happens even if both parents live

in the same village rather than the tundra. I am not going to talk about the carbohydrate diet or about the imperfect schools and education's programs which do not teach about the native people's unique ethnic history. It might be explained as some sort of protection from the pathogenic factors.

Another point which is very important is the long and constant maintaining of children at the child care

institutes without communication with parents, and being away from their ethnic and social environment. This situation could lead to the partial or complete dissatisfaction of the emotional, social and sensory needs of children. As far as I am concerned, that separation might be classified as a deprivation. The unprecedented mass nature of these practices can be excused by nothing. The situation is made more severe by the fact that the teachers and nursing staff are Newcomers.

Certainly, all these factors lead to the deep deformation and delay of physical and psychological development. Practically 100% of Native children are separated from families and go through that conveyor belt system. Is there any other nation in the world like that?



Summer at reindeer herder's camp on Bolshoi Anyui River, Chukotka



Separations of parents from their children inevitably affects the parents too. The tender and delicate attitude and caresses to children have completely disappeared.

The psyche of young men are exposed to negative effects during their military service time. Questionnaire results show that most of these men suffer indignities based on their race. The young natives going through military service are turned away from the native culture, but at the same time do not accept any other culture.

The young people do not bring the average moral personal orientations and precepts to active life, and are not equipped to make independent and responsible actions.

Sometimes, the psychological status of contemporary Native youngsters is considered to be a natural disability, retardation or in the best case as a different way of thinking or different life philosophy. But, in most cases the psychic condition of the Natives is not natural, it is acquired.

The last factor in the formation of indifference is putting up with the farmstead environment. Earlier on, the reindeer and formation of reindeer herds were the center of life for the indigenous reindeer industry - now the creation of reindeer farms are taking away all these previous responsibilities. From being a free worker and owner, a nomad was then made into a laborer; made into an employee making reality out of someone else's wishes, but living an unreal life himself. Many native people already accept this as a necessity, or even as a favor. The psychology of dependence without rights is now predominant.

This happened not only with reindeer herders, but with fishermen, hunters and marine mammal hunters as well. All their lives were to be transformed and limited from outside. And this "new order" destroyed all other systems of "values" and "goals". Everything became "equally meaning" or even "equally unmeaning" (as a professor F. Bacin says). This is considered as a loss of "sense of a life" and is accompanied by powerlessness, worry, and awkwardness.

The wish to get rid of that condition is huge, but how? People cannot understand the reasons for these conditions, and their social and psychological defense forces are weak. Mr. Eric Fromm says that there are two options to get away from that condition. One way is disappearing from the problem environment. The other is the destructive tendency, which usually could be directed to the outside

subject that is a threat. If for some reasons (like a personal weakness) the destructive tendency could not be directed to the outside subject, the people direct it against themselves. The reaching of certain points in those tendencies leads to sicknesses or suicides. This is exactly what is happening with the Native people of the North.

At a certain stage, the Native people were engulfed by an absolutely different type of culture. If they could have had opportunity to go step by step, and adapt to a new culture, it would not have been so hard. But it was not so. They faced the necessity of immediate integration and closeness to the different civilization. The individuals with good tolerance got along well and later assimilated into the new environment. Those who were not compatible detached through disease, suicide and homicides. In the earlier stages the suicides took places only in the so-called "traditional" ways or in the "clear (pure)" cases such as hanging or shooting. Later, the "new culture" brought them new effective instruments such as alcohol.

As I see it, the depression of the Native people is so deep that the suicidal intent and impulse are transferred subconsciously. Trying to overcome that state, using the illusory compensatory meaning of alcohol, a person increases the dosage of alcohol. Sooner or later the alcohol turns out to be a direct or indirect tool of suicide.

According to the analysis of pathological data at the Bilibino hospital, suicide as a cause of death among the native population ranked seventh in 1981-1991. In 1992 it was in ninth place. Alcohol poisoning as a cause of death is taking a stable third

place, and death from alcohol related causes is taking fifth place. If combined, the cases of suicide, along with death from the direct effects of alcohol (poisoning, aspiration) and cases of accidents related with alcohol abuse would take second place as a cause of death and the rate would be a 251 cases per 100,000 population. As far as I understand it, the arsenal of suicide instruments and means is getting larger!

Perhaps the destructive role that pesticides and tracked vehicles play with the environment is paralleled with the indigenous people, who are killing themselves indirectly as well. There might be scientific research to prove or to reject this idea. I do it without scientific claims, but with a hope to find a way to preservation. A lot of people think that the depression is so deep that there is no chance to revive.



Winter at reindeer herder's camp on Moly Anyui River, Chukotka



I think that it is possible to save the situation if some urgent action is undertaken. It is not the financial achievements of farms or workers that is needed to help the Native people. With financial success being a priority, the ethnic traditions suffer, and vice versa. The leaders of farms understand this, and intentionally sabotage and distort the process of giving back the land and reindeer herds. What did they do? For example, they sign contracts with some foreign companies. Do the foreign businessmen know what is happening, without knowing the Native people involved? It is not good to accept decorative actions as a revival of the Native traditions; we are talking about an external, non-Native judgment of what ethnic traditions are, from those who still control the farms.

The main principle of a real revival is the opportunity of free creation of style of life and self-projections of their life. The main condition of revival is creation of necessary economic and judicial guarantees and conditions. One of the inevitable conditions is the temporary keeping of distance or separation between the ethnic communities. The priority actions are to create plenipotentiary legal Native corporations or Committees which are separate and not associated with, or replaceable by, any state or Federal organizations.

These foundations have to be in charge of the land and distribution of those lands where there still exists a traditional style of life. This is a very important issue, because the present stage of recovering and consolidating Native lands is going on in spite of Convention MOT #169. The living surroundings might be destroyed completely,

A special type of management is supposed to be put over the land, as well as a self government and regulation of economy of state as a private one. It must start new cultural programs, as well as education and health care services. The finance and state budget for those programs

must go to the plenipotentiary representative of the Native organizations.

It is necessary to start construction along the paths of traditional migration of nomadic people in order to have elementary hygienic working and living conditions.

At the same time, a small school must be opened at all tundra bases. It is extremely important to be very selective in hiring teachers. Knowledge of native languages is not as important as having knowledge of and teaching about active life. This is a nerve of the revival.

It would be very nice to have, even temporarily, the ethnologists, psychologist and sociologists be teachers. Being both enthusiastic and specialists in these sciences, they could give back an almost-lost potential to the Native people.

The health care system also needs to be seriously improved. At the present moment the tundra people can get only emergency services or treatment at hospitals in the most serious cases. Prevention programs, health education programs and epidemiological information are not available for them. It happened because the health care structure on the North is copied from the Central Russia health care system. The only unit that is really unique, effective and suitable for the North are the Mobile Medical Teams - but they are not independent, and do not have enough equipment and means of communication.

New concepts for a health care system for the North need to be found, like a Medical Center for Native people. The first experience of this concept is intended for Bilibino, Chukotka, but at present all movement is frozen.

The above-mentioned actions are not easy. There may be a lot of obstacles and complications, but they are the only ones giving some hope for revival, as far as I am concerned. All other ways are leading to the assimilation or brightening up or disguise.

# Substance Abuse Problems in the Magadan Region of the Russian Far East

Vladimir P. Lisenko, M.D. <sup>(1)</sup>

Bill Richards, M.D. <sup>(2)</sup>

## INTRODUCTION

There are similarities between Alaska and the Magadan Region of the Russian Far East:

1. Both regions are geographically large territories.
2. The population is about 500,000 in both.
3. There are small villages far from the bigger cities and towns.
4. There are problems with communication and transportation, especially so for the more remote villages.
5. There are indigenous Native populations, which have faced rapid changes in recent years. In the Magadan Region, there are about 15,000 Natives - Chukchis, Eskimos, Evenks, Kamchadals, Inrkuts, and some other small nations. Compared to Alaska Natives, the Natives in the Magadan Region are more of a minority in terms of percent of the total population.
6. There are high rates of alcohol-related problems in both locations.

Because of these similarities, this report summarizes what is known about substance abuse problems and services in the Magadan Region, which may be of interest to Alaskans.

## EXTENT OF SUBSTANCE ABUSE IN THE MAGADAN REGION

Magadan, compared to other regions of Russia, has the highest rate of alcohol abuse. There are very high rates for alcohol-related injuries, poisonings, suicides, homicides, and alcohol psychoses. Chart 1 shows that 40-60% of all deaths in the Region are the result of accidents, intentionally inflicted injuries thought to be alcohol-related, or from the direct effects of alcohol. The rates are even higher for Natives than for "newcomers" to the Region which results in low Native life expectancies.

Drug abuse is a smaller problem than alcohol abuse. There is a fair amount of marijuana use, plus use of

benzene (gasoline) by younger children as an inhalant, and the use of alcohol substitutes when alcohol is not readily available.

## REASONS FOR THE HIGH INCIDENCE OF SUBSTANCE ABUSE

There are many suggested reasons for the high rate of substance abuse in the Magadan Region. Some reasons are social and some are biological.

Socially there are severe work conditions and difficult living conditions within the Region. There is a lack of skilled jobs for most of the population. For Native peoples, the man often has to live separately from the rest of his family. Since he may be working out in the tundra as a reindeer herder far from the village where his wife stays, and the children may be in a State Boarding School in a bigger and different community from where the mother lives, a result is a separated family. These are only a few selected concerns, and alone they can't explain a high incidence of substance abuse.

Biologically, it may be that Native peoples have no resistance to alcohol. Therefore, the person is not protected well against alcoholism, and the disease becomes prominent quickly.

Many women do not stop drinking when pregnant. High rates of Fetal Alcohol Syndrome and Central Nervous System Diseases related to Alcohol Birth Defects are suspected.

It appears that the level of education for Native children in some villages is declining, and children are not as educated as children their same age were 10-15 years ago. The adults have lower life expectancies than some years ago. For example, in some villages there are more females than males because the men, 25-40 years old, have predominantly died from alcohol poisoning, suicide, alcohol-related drowning or freezing to death.

## TREATMENT STRATEGIES

Those who consider it unacceptable to drink alcohol understand the need for education and communication

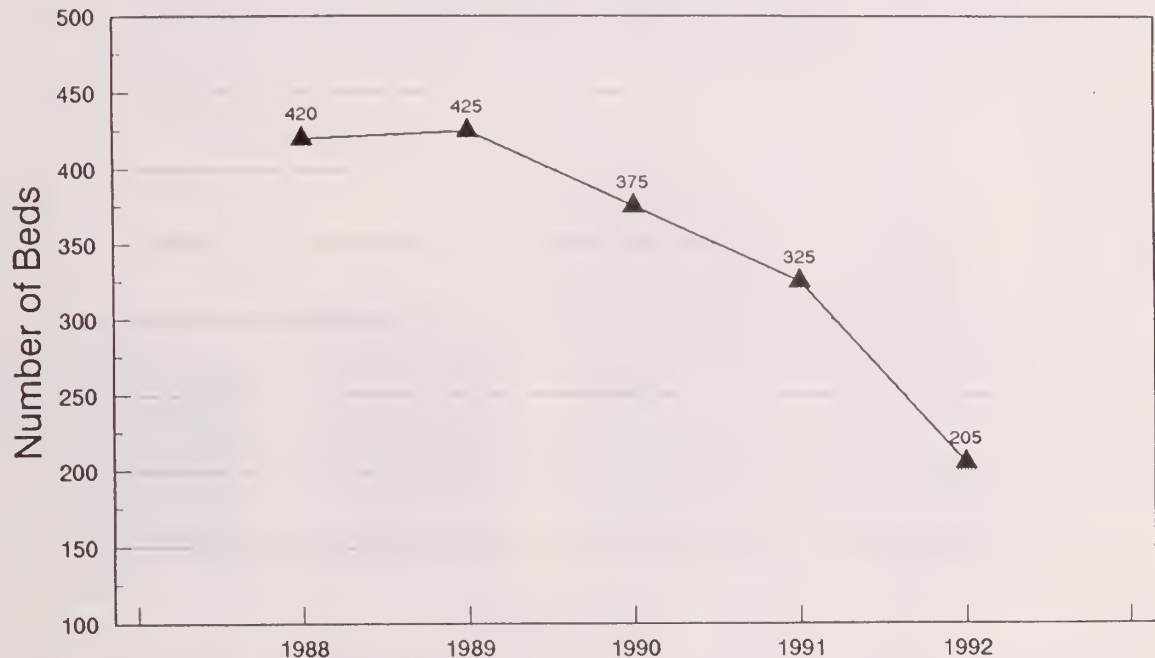
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## NARCOLOGY BEDS IN THE MAGADAN REGION

1988 - 1992



Source: Reference Manual Showing Narcological Information for the Population of Magadan Oblast for the Year 1989-90. City of Magadan 1991 p7

which can lead to the reconstruction of native cultures. There is discussion of the desire to return to traditional native occupations and life styles, to bring back traditional native holidays, to touch the heart of the people who need to get involved in a life without alcohol. Alcoholics Anonymous (AA) has recently been introduced in Magadan, Sireniki and Konergino. Some of these groups are trying to get community members involved. About six to ten villages currently have started AA.

In some villages distant to the bigger villages, some traditional treatments may still be practiced. Special hypnotic treatments are also used for alcohol problems. Programs for teenagers are starting, but teen problems, with more poly-substance abuse are more acute.

There was a special Alcohol Reform Law (ARL) passed nationally throughout Russia in 1985. This law made it difficult to legally purchase alcohol. Many teenagers then tried to use a variety of substitutes for alcohol, including gasoline vapors, benzene and even mosquito repellent. In some villages, the teenagers asked their grandparents to tell them about traditional mushrooms and plants with hallucinogenic qualities, and they began to use those. There was a sharp increase in deaths in the 1-2 years following passage of the ARL. Those were related to hypothermia and suicides following the frustrations of boy-girl relationships.

People from villages resist going to distant big cities for treatment in special clinics. They don't feel comfortable there, so there have been attempts to get treatment facilities where people live. There are court-ordered treatments for alcoholism. Facilities, however, are not ideal. Preventive education has just begun, i.e., media coverage on TV regarding FAS and the influence of alcohol on one's personal health.

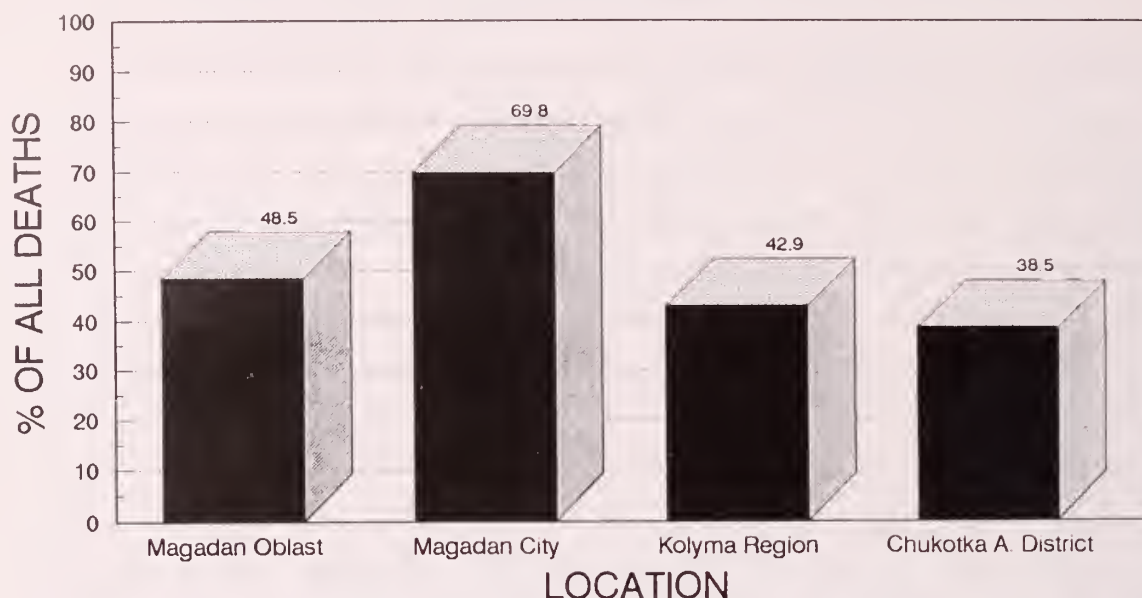
The economic situation is very difficult and potentially severe for the people who live in the Northern part of the country. It is more expensive to live there, since weather conditions are harsher and food has to be imported. A law to get special help for Northern peoples is in the process of development within the government.

In spite of all the problems, there continue to be success stories. People feel responsible, and see that people can recover. In Konergino, there is a woman with a good position in the village. She had volunteered to get treatment, was successfully treated, and therefore was a positive role-model to others.

During follow-up, one year later, she described the effects her recovery was having on others. She said that the people around her looked at her life very attentively, to see whether it was working or not. They wanted to see what would happen as a result of her treatment, and each



## PERCENT OF TOTAL DEATHS FOR 1989-90 FROM SUICIDES, ACCIDENTS, AND OTHER DEATHS PROBABLY ASSOCIATED WITH SUBSTANCE ABUSE



Source: Reference Manual Showing Narcological Information for the Population of Magadan Oblast for the Year 1989-90, City of Magadan. 1991 p77

of her steps were analyzed by the people around her. Shortly after her treatment for alcohol problems, she developed spinal problems. Some people in the village told her "when you were drinking, you felt good. But now after treatment you have some problems!" She soon physically recovered from those spinal problems. Children like to come to her house, and tell everybody in the village that she had a clean, warm and nice house, and that her whole family got together in the evenings - the children from school and the parents from work. This woman serves as a good example for other families in the villages. She is an active member of an AA program. People's opinions can be turned around; everybody can see how treatment has changed the lives of this woman's family.

### SUMMARY

People in the Magadan Region are hopeful that citizens of Alaska will make contact with Native peoples, especially in the Magadan region. Right now, rates of alcohol-related problems are high and economic difficulties are leading to cutbacks in existing programs (see Chart 2).

Alaskans have much good experience and information to share, and there is a lot that can be accomplished,

as illustrated by the story of the recovering woman, and her influence upon people by example.

Dynamics of the morbidity of alcoholism and alcoholic psychosis in regions of Siberia and the Far East.

Chart 3 shows dramatic rises in alcoholism/alcoholic psychosis rates between 1965 and 1985.

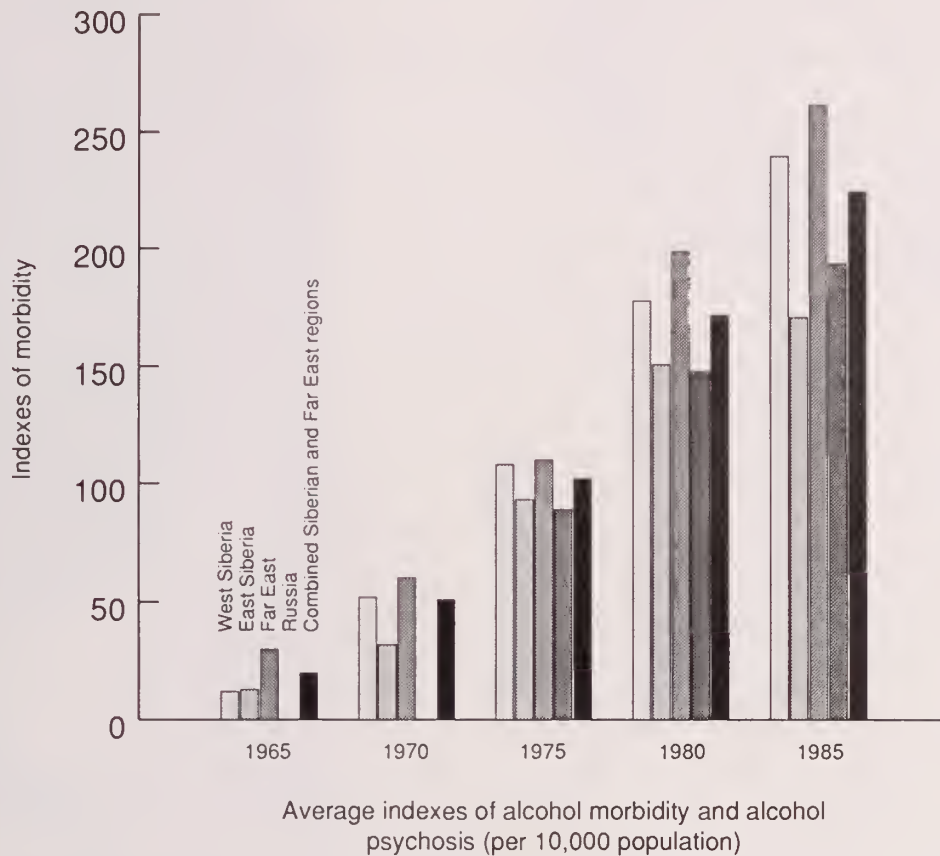
On the scale used, rates were about 20 per 10,000 in 1965, and in 1985 were about 250 per 10,000. Rates appear to be higher in the Far East, to be second highest in Western Siberia, and to be slightly lower in Eastern Siberia.

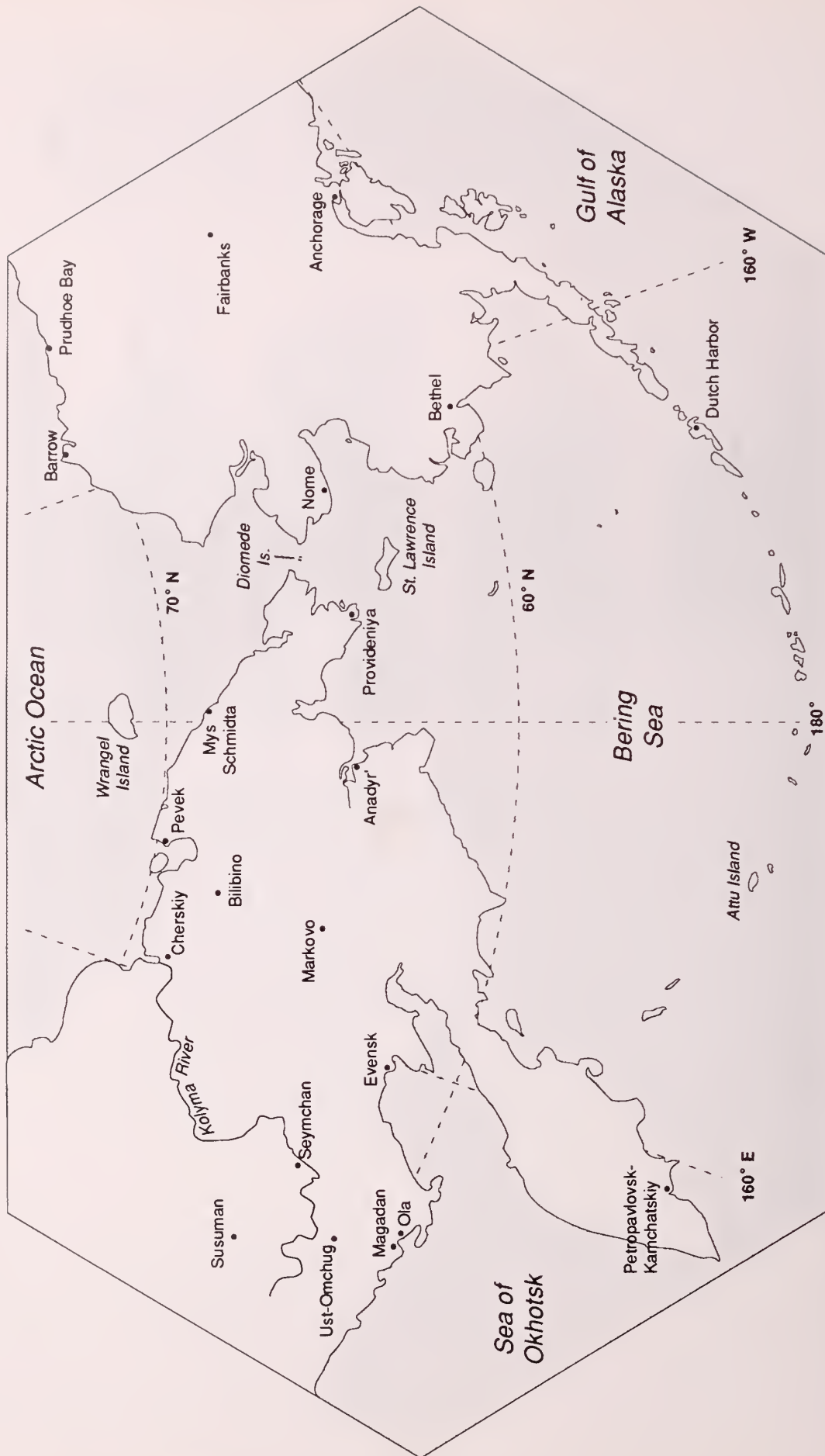
Rates for Russia as a whole seem to have followed the same general trend towards rising, but are somewhat lower than the rates given for Siberia and the Soviet Far East.

Two questions arise out of this data:

1. Why the increase since 1965?
2. Why worse in the Far East?

# Dynamics of alcoholism morbidity and alcohol psychosis in the Siberian and Far East regions of Russia.







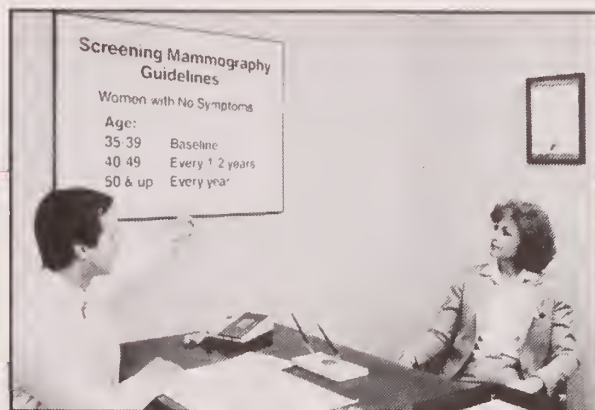


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Volume 36, Number 4

October/November/December 1994



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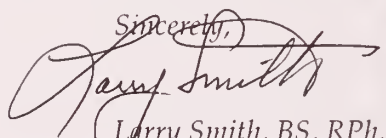


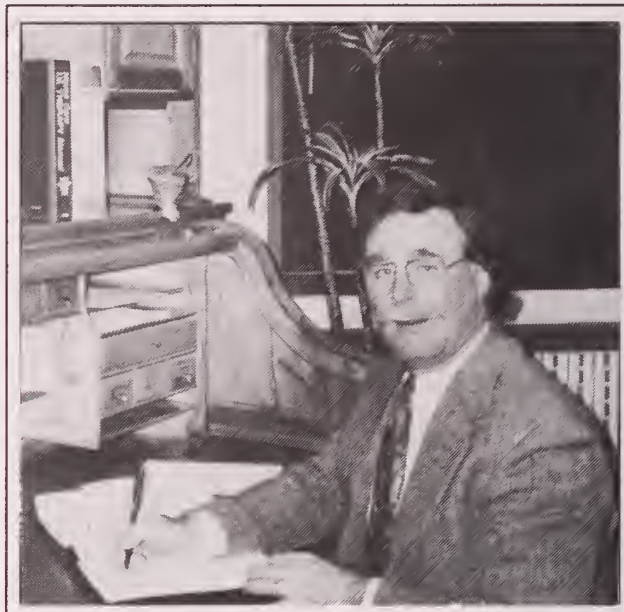
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# Interleukin-2 Receptor Beta and CD57 Expression in Orbital Tissues from Patients with Chronic, Stable Thyroid-Associated Ophthalmopathy

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## SUMMARY

In order to determine whether components of the interleukin-2 receptor (IL-2R) and lymphoid cells are present in extra ocular and periocular tissues from patients with chronic, stable thyroid-associated ophthalmopathy (TAO) we studied 16 specimens of extra ocular muscle and periorbital connective tissue from 14 patients with chronic, stable, TAO using an immunohistochemical assay and a panel of murine monoclonal antibodies reactive with IL-2R alpha and beta components and lymphoid cell surface markers. As controls we studied orbital tissues from 11 patients undergoing surgery for unrelated orbital disorders. All extra ocular muscle specimens from patients with TAO exhibited IL-2R beta expression primarily on the perimysium and endomysium surrounding the ocular muscle fasciculi and fibers of which nine specimens stained intensely. The Natural Killer (NK) cell marker CD57

was the most common cell surface antigen detected, in seven of nine specimens, whose localization often corresponded to that of IL-2R beta distribution. No IL-2R alpha expression was detected in any specimen. Seven of the 11 control specimens were positive for IL-2R beta but in a less intense fashion than in TAO specimens while no CD57 staining was detected. T cell, B cell, and cells of granulocyte and monocyte lineage were only occasionally found in both TAO and control specimens. The aberrant expression of IL-2R beta and CD57 which may be representative of NK cell presence in extra ocular muscle tissues from patients with stable, chronic TAO may play a role in the pathogenesis of the ophthalmopathy.

Despite a great deal of clinical experience and a major research focus by many groups thyroid-associated ophthalmopathy (TAO) remains a poorly understood disease (1). A possible role of cytokines produced at the site of the auto immune reactions in the orbit has been proposed by some workers (2,3). The lymphokine IL-2 specifically interacts with the interleukin-2 receptor (IL-2R) (4) and affects the growth and differentiation of T cells, B cells, natural killer cells (NK), glioma cells, and cells of the monocyte lineage. The IL-2R comprises at least two distinct subunits namely, alpha (a 55 kD protein) and beta chains (a 70/75 kD protein), and exists in three isoforms with high (dissociation constant  $K_d$  is  $10^{-11}$ M), intermediate ( $K_d$   $10^{-9}$ M), and low ( $K_d$   $10^{-8}$ M) affinity for IL-2 (3). The high and intermediate affinity receptors, which are thought to comprise the alpha and beta heterodimers, and the beta chain alone, respectively, are effective in IL-2 mediated signal transduction. Since the low affinity receptor, which consists of the alpha chain alone is ineffective, the beta but not the alpha chain must be indispensable (4-6). The beta chain has an intermediate affinity for IL-2 binding

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and transduces intracellular signals when expressed on lymphoid and oligodendrogloma cells. The beta chain is not functional when it is expressed on fibroblast cells (6). A third IL-2 receptor subunit, a 64 kD gamma chain, which has been identified by Takeshita (5) seems to play a crucial role in facilitating IL-2 binding by IL-2R beta and in receptor signaling.

Most resting T cells, B cells, NK cells and monocytes do not express IL-2R alpha, although they can be induced to do so by a variety of stimuli (4). Most NK cells constitutively express IL-2R beta and IL-2R gamma (4). Natural Killer cells are a subset of lymphocytes found in blood and lymphoid tissues which possess the ability to kill certain tumor cells and normal cells infected by virus. This killing is not restricted by major histocompatibility components. Addition of IL-2 to NK cells enhances their activity and can lead to the generation of so called lymphokine activated killer (LAK) cells and IL-2R alpha expression. LAK cells demonstrate enhanced cytolytic capacity and a very broad target specificity, killing a wide variety of tumor and normal cells, including epithelial cells (4). In order to mediate immune responses T cells must change from a resting to an activated state. Typically, T cells are stimulated by foreign antigens initiating a series of cellular activation steps leading to production of IL-2. The high affinity IL-2R is also expressed during T cell activation. Interaction of IL-2 with its induced cellular receptors triggers cellular proliferation, culminating in the emergence of mature effector T cells required for the full expression of the immune response (4). We now report immunohistochemical evidence for the presence of increased IL-2R beta and CD57 expression in orbital tissues from patients with chronic, stable TAO.

## MATERIALS AND METHODS

### Tissue Specimens

Tissue was obtained from 14 patients with TAO in the context of attempts to improve their vision and appearance. Orbital tissue was also obtained from 11 patients with strabismus, blepharoptosis or fat prolapse as controls. Our use of tissue did not in any way impinge on diagnosis or treatment. The tissues were placed into sterile, moist gauze in the operating room and fixed within three hours.

Tissue was fixed in Tago (Tago Inc., Burlingame, CA) (14 cases) or Zenkers (15 cases), embedded in paraffin, cut into 4 um sections and placed onto glass slides. All antibody reactions were performed at room temperature. Non specific binding was blocked with 2 percent nonfat dry milk powder in phosphate buffered saline (PBS). The panel of mouse monoclonal antibodies (MCAB) used were; anti-human IL-2R beta (Coulter

Immunology, Hialeh, FL), anti-human IL-2R-alpha (BioSource International, Camarillo, CA), anti-human IgM (Boehringer Mannheim, West Germany), anti-human CD56 (7) (Natural Killer cells, Coulter Immunology, Hialeh, FL), anti-human CD57 (8) (expressed on 60 percent of active Natural Killer cells, neuronal tissue, and a subset of T cells, Biodesign, Kennebunkport, ME), anti-human CD2 (9) (expressed on all T cells, DAKO, Carpinteria, CA), anti-human LeuMI/CD15 (10) (granulocytes, monocyte precursors, Becton Dickinson, San Jose, CA), and anti-human CD23 (11) (activated B cells; the low affinity receptor for IgE, Coulter Immunology, Hialeh, FL). MCAB were diluted in PBS to a final concentration of 10 ug/ml and incubated with deparaffinized tissue sections for two hours at room temperature. Bound primary antibody was visualized using a biotin/streptavidin staining system (San Ramona, CA). Second antibody was an anti-mouse IgG antiserum conjugated to alkaline phosphatase. Levamisole (125 mM) was added to the final chromagen mixture to ensure inhibition of endogenous alkaline phosphatase activity thereby reducing background staining. Tissues were washed twice in a gently agitating PBS bath for 10 minutes between each step. Finally, all sections were counterstained with Meyer's hematoxylin and mounted in "glycergel" (DAKO, Carpinteria, CA).

## CASE REPORTS

### Patients with Thyroid-Associated Ophthalmopathy

**Case 1:** A 54-year-old white woman with mild TAO and an otherwise normal neuro-ophthalmic examination underwent a muellerectomy for right upper lid retraction. She was taking no medications.

**Case 2:** A 30-year-old white man presented with marked bilateral exophthalmos and upper lid retraction with an otherwise normal neuro-ophthalmic examination. He underwent a three wall orbital decompression on the left side and a four wall orbital decompression. Medicine included thyroxine for hypothyroidism.

**Case 3:** A 49-year-old white woman presented with marked bilateral exophthalmos causing exposure keratopathy and bilateral upper lid retraction. Medications included thyroxine. Neuro-ophthalmic examination carried out prior to a right three-wall orbital decompression and a left four-wall orbital decompression revealed enlarged blind spots. B scan ultrasonography showed enlarged extra ocular muscles. Postoperatively she was treated with oral prednisone in reducing doses. Approximately two years later a left medial rectus recession on an



adjustable suture was performed with a left and right Mueller's excision of both upper lids.

**Case 4:** A 47-year-old man previously treated with radioactive iodine for hyperthyroidism, presented with diplopia and congestive TAO. He was treated with a short course of oral prednisone and maintained on propyl thiouracil (PTU) for his hyperthyroidism. He received bilateral orbital radiation treatment with 2520 cGy in 14 fractions over 20 days and 10 mg prednisone per day for refractory congestive TAO. Six months later bilateral inferior and medial orbital wall decompressions were performed for compressive optic neuropathies. Subsequently left inferior rectus and medial rectus recessions on adjustable sutures were performed for diplopia. Finally, this patient underwent additional right inferior rectus and medial rectus recessions on adjustable sutures.

**Case 5:** A 48-year-old white woman presented with TAO and diplopia due to bilaterally contracted medial recti. Medications included Inderal and Tapazole. Bilateral medial recti recessions on adjustable sutures were performed for an esotropia. She received no prior treatment with radiation or steroids.

**Case 6:** A 45-year-old white woman presented with diplopia on primary gaze. Medicines included Inderal and Provera. She was treated with radioactive iodine for Graves' hyperthyroidism and underwent radiation treatment with 2520 cGy bilaterally for congestive TAO. Approximately 10 months later she underwent upper and lower lid blepharoplasties with orbital fat removal and a left upper to lower tarsal transplant for right lower lid retraction for exposure keratitis.

**Case 7:** A 67-year-old white woman presented with diplopia from a contracted right inferior rectus muscle with right lower lid retraction and underwent a right inferior rectus recession on an adjustable suture. Medicines included thyroxin, Quinaglute, and Lanoxin.

**Case 8:** An 84-year-old white woman with TAO, previously treated with radioactive iodine presented with diplopia. Medicines included thyroxin and Lanoxin. A left inferior rectus recession on an adjustable suture was performed.

**Case 9:** A 52-year-old white woman presented with diplopia and was diagnosed with severe, acute TAO. She was prednisone intolerant and received

radiation therapy for the acute inflammatory phase. A left medial rectus recession on an adjustable suture was performed eight months later.

**Case 10:** A 52-year-old black woman taking thyroxin for hypothyroidism presented with diplopia and a compressive optic neuropathy. Oral prednisone was begun which relieved her optic neuropathy but not her diplopia. She underwent a right inferior rectus recession on an adjustable suture. One year later a left medial and inferior orbital wall decompression was performed followed by a left medial rectus recession on an adjustable suture after another 12 months.

**Case 11:** A 65-year-old white man presented with chronic TAO and diplopia secondary to left inferior rectus contracture. He underwent a left inferior rectus recession on an adjustable suture.

**Case 12:** A 44-year-old white woman presented with TAO and bilateral optic nerve compression. She had previously received radioactive iodine for hyperthyroidism as well as oral prednisone, radiation treatment to both orbits, and bilateral orbital decompressions for severe TAO. A repeat bilateral lateral and inferior wall decompression was performed to augment the orbital decompression.

**Case 13:** A 52-year-old white woman with a history of moderately active TAO for approximately 14 years underwent a right two-wall decompression for compressive optic neuropathy. Approximately nine months later a second two-wall decompression was performed for recurrent compressive optic neuropathy.

**Case 14:** A 46-year-old white woman underwent bilateral Mueller's excisions for upper lid retraction. TAO had been stable and inactive for three years. She was euthyroid following radioactive iodine treatment two years prior to the operative procedure.

## RESULTS

Results of immunohistochemical analyses of orbital tissue from patients with chronic, stable TAO and as controls, from patients with non immunological orbital disorders using MCAB reactive with 1L-2R beta and alpha and various lymphoid cell markers are summarized in Table 1 and examples shown in Figure 1. An inherent problem with immunohistochemistry is that it is often impossible to accurately quantitate staining reactivity. However, an attempt at quantitating staining

**Table 1. Interleukin-2 Receptor expression and lymphoid cell infiltration in orbital tissues from patients with Thyroid-Associated Ophthalmopathy**

Case #	Age/Sex	Tissue	IL2Rbeta	IL2Ralpha	IgM	CD23	CD56	CD2	CD15	CD57
<b>TAO</b>										
1.	54/F	Mueller's	1+*	0	0	NT	O	NT	2+	NT
2.	30/M	Orbital Fat	1+	0	0	NT	0	NT	NT	NT
3.	54/F	Medial Rectus	2+	0	0	NT	NT	NT	0	NT
4.	47/M	Medial Rectus	1+	0	0	0	0	0	0	0
5.	47/M	Medial Rectus	1+	0	0	0	3+	0	0	2+
6.	48/M	Medial Rectus	3+	0	0	0	0	0	0	3+
7.	45/F	Medial Rectus	3+	NT	0	0	0	NT	NT	NT
8.	67/F	Inferior Rectus	3+	NT	0	0	NT	NT	NT	NT
9.	84/F	Inferior Rectus	3+	0	0	NT	0	0	1+	3+
10.	52/F	Medial Rectus	3+	0	0	0	0	0	0	1+
11.	52/F	Medial Rectus	3+	0	0	0	NT	0	1+	2+
12.	52/F	Medial Rectus	1+	0	0	0	0	0	0	0
13.	65/M	Inferior Rectus	3+	NT	0	0	NT	NT	NT	NT
14.	44/F	Lateral Rectus	3+	NT	0	0	NT	NT	NT	NT
15.	52/F	Medial Rectus	3+	0	0	2+	0	0	3+	1+
16.	46/F	Mueller's	1+	0	0	0	2+	0	0	1+
<b>Control</b>										
1.	45/M	Inferior Oblique	0	0	0	0	NT	NT	NT	NT
2.	54/M	Lateral Rectus	1+	0	0	0	0	0	0	0
3.	54/M	Lateral Rectus	0	0	0	NT	NT	NT	0	NT
4.	72/F	Orbital Fat	1+	0	0	0	0	0	0	0
5.	72/F	Orbital Fat	1+	0	0	0	0	0	0	NT
6.	65/F	Inferior Oblique	1+	0	0	NT	0	NT	NT	NT
7.	29/M	Medial Rectus	1+	0	0	NT	0	0	NT	0
8.	81/F	Lateral Rectus	0	NT	0	NT	NT	NT	NT	NT
9.	68/F	Mueller's	0	0	0	0	0	0	1+	
10.	70/F	Medial Rectus	2+	0	0	NT	NT	NT	0	NT
11.	64/F	Lateral Rectus	0	0	0	NT	NT	NT	0	NT
12.	10/M	Mueller's	0	0	0	NT	NT	NT	0	NT
13.	41/M	Lateral Rectus	2+	0	0	NT	0	NT	NT	0

\*Specimen staining reactivity key: 0 no staining; 1+ mild staining; 2+ moderate staining; 3+ intense staining  
NT = Not Tested

was made by assigning O to no staining reactivity, 1+ to mild staining reactivity, 2+ to moderate staining reactivity, and 3+ to intense staining reactivity. Staining reactivity refers to a combination of the amount of cells or tissue stained and the level of color generated within the specimen. Reactivity with anti-human IL-2R beta was demonstrated in the perimysial and endomysial connective tissue surrounding the muscle tissue, respectively in all 16 specimens from 14 patients with TAO; intense staining with anti-human IL-2R beta was observed in nine specimens (Figure 1, B). Staining for the NK cell marker anti-CD57 was observed around the muscle fascicles in seven out of nine TAO specimens, which seemed to correspond to the IL-2R beta distribu-

tion in seven cases (Figure 1, D). Only two of 11 TAO specimens stained with the NK cell marker anti-CD56. Staining with the granulocyte/myeloid marker anti-LeuM1 (CD15) was found in four of 11 TAO specimens. Two out of 16 TAO specimens stained with anti-human IgM primarily on the perimysium and surrounding fibrous connective tissue. Two out of 10 TAO specimens stained with the pan T cell marker anti-CD2. One out of 12 specimens from patients with TAO examined stained with the B cell activation marker anti-CD23. No staining occurred with anti-human IL-2R alpha when reacted with TAO (Figure 1, C) or control (Figure 1, G) specimens and may have been secondary to antigen disruption as a result of the paraffin embedding technique.



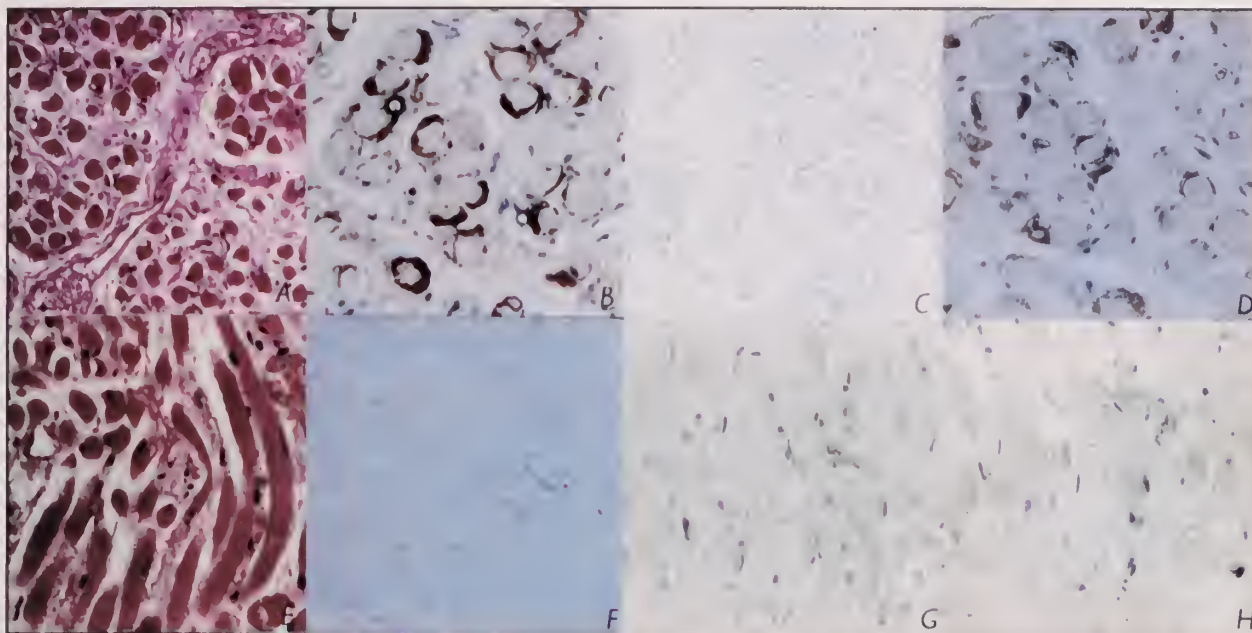


Figure 1.

Representing staining of ocular tissues from patients with TAO and control subjects with MCAB reactive with IL-2R beta and various lymphoid cell markers. The protocol for immunohistochemical staining of orbital tissues, the MCAB used and incubation details are described in Materials and Methods. Specimens (A-D) are of medial rectus muscle obtained from TAO Case 6: A, Hematoxylin and Eosin stain, magnification X160; B, reaction with anti-IL-2R beta MCAB, magnification X160; C, reaction with anti-IL-2R alpha MCAB, magnification X160; D, reaction with anti-CD57 (NK cell) MCAB, magnification X160. Control specimens (E-H) are of inferior oblique muscle from Case 6: E, Hematoxylin and Eosin stain, magnification X160; F, reaction with anti-IL-2R beta MCAB, magnification X160; G, reaction with anti-IL-2R alpha MCAB, magnification X160; H, reaction with anti-CD57 MCAB, magnification X160.

Fixation in either Tago or Zenkers did not seem to matter. BioSource International recommended using frozen sections as positive controls, unfortunately not enough specimen was available. IL-2R beta was demonstrated in seven out of 13 control specimens, although no specimens stained intensely and only two specimens stained moderately (Figure 1,F).

Of the 13 control specimens no staining occurred with anti-human IgM. No staining occurred with anti-CD23, anti-CD56, anti-CD2, or anti-CD57 (Figure 1,H) in the few specimens tested. Comparisons between Zenkers and Tago fixatives did not reveal significant differences in reactivity with any MCAB for either TAO or control specimens.

## DISCUSSION

Orbital tissue specimens from patients with TAO were probed with MCAB reactive with lymphoid cell surface markers and the IL-2R alpha and beta components. All muscle specimens from patients with TAO exhibited IL-2R beta expression mainly on the perimy-

sium and endomysium surrounding the ocular muscle fascicles and fibers, respectively, which was intense in over half of the specimens examined. CD57 was the most consistent cell surface marker detected. Although CD57 and CD56 are both NK cell markers, CD57 recognizes about 60 percent of active NK cells and a subset of T cells and is thought to be involved in cellular adhesion events (10). CD57 has a broader expression (6) than CD56 perhaps accounting for its greater expression in TAO patients tissues. Control specimens reacted, but in a less intense fashion for IL-2R beta while no CD57 or CD56 cell surface markers were detected. Cells of T cell, B cell, and cells of granulocyte and monocyte lineage were only occasionally found in either TAO or control specimens. IL-2R beta expression was often prominent in control specimens from patients who had sustained severe traumatic injuries such as ruptured globes or phthisis, suggesting that IL-2R beta is induced and expressed in association with any form of inflammation.

The IL-2/IL-2R system, which initiates and activates T cell, B cell, and NK cell responses (12) may play a



role in the pathogenesis of TAO. Indeed IL-2R and its ligand have been implicated in many autoimmune disorders (4). Modulation of the IL-2/IL-2R system by cyclosporin, FK506, and rapamycin which act by suppressing the immune system, has revolutionized organ transplantation (4). In a randomized prospective trial, there was a greater reduction of early renal allograft rejection episodes in human recipients of cadaver donor renal allografts who were treated with anti-IL-2R alpha MCAB in addition to standard immunosuppression, compared to those receiving conventional therapy alone (4). Krusel and colleagues (13) examined the immunosuppressive effect of ART-18, a MCAB reactive with the IL-2R in corneal graft rejection experiments in a rat model; suppression of graft rejection was obtained with subconjunctival administration of ART-18 while administration of antibody systemically produced no significant effect.

Tallstedt (14) studied frozen cryostat sectioned biopsies from five patients with Graves' disease. In four cases weak cellular reactions with only a few lymphocytes of B and T cell origin were noted and there were no signs of muscle cell damage. Interestingly, an anti-IL-2R antibody was included in the panel of MCAB used although the results were not specified. Weetman (15) examined paraffin embedded specimens from three patients with TAO and found predominantly T cells and, to a lesser extent B cells. Differences compared to our results may be due to the fact that specimens were obtained from one patient with active TAO who suffered trauma resulting in enucleation, while disease activity was not stated for the other two patients. In addition trypsinization was used on specimens prior to testing for T cells which may have damaged cellular proteins.

The presence of IL-2R on fibroblasts and endomysial connective tissue cells may not be functionally significant since IL-2R beta bearing fibroblasts do not transduce or activate intracellular signals when bound with IL-2 (4). On the other hand the IL-2R found on NK cells may be significant since NK cells, once activated with IL-2 attached to beta, and possible gamma receptors could possibly induce intracellular internalization and activation to LAK cells. The claim by Tallstedt (14) and Hufnagel (16) that a well preserved skeletal architecture is present and that primary muscle damage does not occur, at least in the early stages can only be confirmed by studying orbital tissues from patients with very early ophthalmopathy. The fact that IL-2R beta and the CD57 marker reminiscent of NK cell expression is found in specimens from chronic, stable TAO and not in control suggests persistent immune activity.

This is the first report demonstrating the presence of IL-2R beta in and around extra ocular muscle specimens from patients with stable, chronic TAO in association with CD57 cell surface expression. This preliminary

observation suggests that investigation of the significance of this aberrant expression and its possible role in the development of the autoimmune reaction in TAO will be fruitful. If confirmed, the IL-2R may be a good candidate for a future therapeutic approach to TAO.

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(continued on page 207)

# Physician Assistant Training for Native Alaskan Community Health Aides: The MEDEX Northwest Experience

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## ABSTRACT

**Background** - From 1980 through 1990, 16 Native Alaskan Community Health Aides and 21 non-Native Alaskans began physician assistant training at MEDEX Northwest at the University of Washington. This study was done to assess the outcome of training Native Alaskan health workers as physician assistants, specifically whether Native Alaskan graduates are working in settings that serve Alaska Natives.

**Methods** - The backgrounds, educational experiences and deployment locations of Native and non-Native Alaskans accepted for training were compared using MEDEX Northwest student records. The 1991 graduate survey was used to compare differences in practice setting, specialty and salary between Native and non-Native graduates working in Alaska in 1991.

**Results** - All of the non-Natives and 81% of the Natives completed the program. Of those completing the program, 100% of the Natives returned to Alaska where 91% found work as primary care physician assistants in clinics serving predominantly Native communities. By comparison 78% of the non-Native graduates returned to Alaska to work as physician assistants, 60% of them in primary care and 15% of them in predominantly Native communities. There were no significant

differences in salary or benefits between Native and non-Native graduates.

**Conclusions** - Physician assistant training for entry level health workers is a viable strategy for increasing the number of under-represented minorities in the health professions. The Native graduates of MEDEX Northwest are returning to communities where they serve Native people both as health care providers and as professional role models.

## INTRODUCTION

There are many geographical and cultural barriers to access to health care for rural Native Alaskans. In villages with populations under 500, people with medical conditions requiring diagnostic or therapeutic skills above the level provided by the local Community Health Aide (CHA) must usually fly to a regional or sub-regional medical center staffed by physicians, physician assistants or nurse practitioners who are able to either treat or refer on to a hospital.<sup>(1)</sup> In addition, the extreme physical environment often makes travel impossible for many days at a time, posing additional risks when emergency evacuation is necessary.

The overwhelming majority of physicians, nurses, physician assistants and nurse practitioners working in facilities that serve rural Natives are Caucasian. These providers often have limited personal support systems within the Native community and are consequently at significant risk for burnout. The resulting high turnover of health providers affects the stability of supervision and support that can be provided to the Community Health Aides in the villages,<sup>(2)</sup> and seriously interferes with continuity of care for Alaska Natives in the sub regional centers.<sup>(3)</sup> At the same time, there are limited opportunities for Native community residents to receive health care professional training. The vast distances in Alaska make availability of prerequisite college courses

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a major challenge to people in the villages, and Alaska Natives who leave the village to attend college in Fairbanks or Anchorage face heavy odds against completing the requirements to go on to careers in medicine or nursing.<sup>(4)</sup>

In 1980, MEDEX Northwest, the physician assistant program at the University of Washington School of Public Health and Community Medicine, began an effort to recruit Alaskans of all ethnic backgrounds for physician assistant training. The only prerequisites were college level courses in English, Anatomy and Physiology, and at least two years experience in a health care field. From 1980 through 1990 a total of 37 Alaskans were accepted into the MEDEX program. Of these, 16 were Native Community Health Aides and 21 were non-Native Alaskans.

The Alaska Community Health Aide Program began in the 1950s as a network of village volunteers who distributed medications to tuberculosis patients. Over the years the range of tasks allocated to CHAs has been expanded to include a wide range of clinical, diagnostic and therapeutic skills.<sup>(5)</sup> CHAs currently receive up to 16 weeks of formal training, in four segments, between which they work in the village clinic. They use a highly structured system of clinical algorithms in their CHA Manual, and rely heavily on daily telephone and radio communication with their clinical supervisors who are MDs, physician assistants, or nurse practitioners in the regional and sub-regional centers.<sup>(6)(7)</sup> Although CHAs are rarely given an opportunity for career advancement outside the CHA program in Alaska, MEDEX faculty believed that they represented an ideal applicant pool for advanced clinical training.

This study addressed three basic research questions:

1. What is the experience of Native and non-Native Alaskans in the MEDEX Northwest physician assistant training program?
2. Do Native Alaskan CHAs trained as physician assistants locate in practice settings that are likely to improve access to care for rural Native Alaskans, i.e. do they meet needs that are not met by non-Native graduates of the same program?
3. What differences are there in the professional environments of Native and non-Native Alaskan graduates of MEDEX Northwest?

## METHODS

**Data.** The data for this study came from two sources.

1. MEDEX student files were the source of infor-

mation on the students' prior professional background, their geographical origin and ethnicity. These data were used to track the training experience of the Alaskan students who entered the program. All students who entered the training program from Alaska, or who were working in Alaska in January, 1991 were included.

2. The second source of data for this study was the 1991 MEDEX graduate survey. This 4-page questionnaire was sent to all graduates of MEDEX Northwest for whom addresses could be ascertained. The survey consisted of 40 questions pertaining to current practice location, specialty, relationship with physician sponsor, frequency of performing certain common clinical tasks, certification status, level of salary and job related benefits. Questions were either multiple choice, yes/no, or Likert scale. The questionnaire was developed and tested in the fall of 1990, and mailed in March, 1991. A second mailing was sent to the non-respondents.

**Measurements.** To compare the experiences of Native and non-Native physician assistant students from Alaska we used information obtained primarily from MEDEX student records. Students were classified as Native if they were, at least, part Aleut, Eskimo (Yupik or Inupiat), or Indian (Athabaskan or Southeast Alaskan). All the non-Native Alaskan students were Caucasian. Using student records, we compared Natives and non-Natives in terms of prior professional background and rates of completion of MEDEX training. Information on location and size of the primary community in which the student was raised was obtained from the graduate survey.

Information on practice location and practice experiences of Alaskan graduates was obtained chiefly from the 1990 graduate survey. MEDEX records were used to identify location of practice for graduates who did not return a survey. Practice location was characterized as rural or urban. Graduates practicing in Anchorage or Fairbanks were considered urban, all others were considered rural. To characterize medical practice experiences of Natives and non-Natives several aspects of practice were examined. Specialty of practice was classified as either primary care (Family Practice, Internal Medicine, Pediatrics, Emergency Medicine and Obstetrics and Gynecology) or specialty practice. We attempted to compare the scope of clinical practice between the two groups by asking graduates to estimate how many times per month they performed a wide range of specific clinical tasks. Salaries and benefits of Native graduates were compared with those of non-Native graduates who entered the program after 1980 because of the tendency for PA salaries to rise with the length of time in practice. The low number of graduates studied prevented further stratification of the salaries by years in practice, specialty, or location.



**Statistics.** The analysis presented here compares the educational and profession experience of Native to non-Native students, both in terms of successful completion of the program, and for those responding to the graduate survey, in their 1992 employment setting. Statistics were confined to Chi-square testing for dichotomous outcome variables and T-tests when variables were continuous. Significant differences at the 95% confidence level are identified in the text and tables.

## RESULTS

**Numbers.** Of the 16 Natives accepted into the program from 1980 through 1990, a total of 13 graduated, 10 of whom finished in time to participate in the 1991 graduate survey. The survey response rate for Natives was 70%. Of the 21 entering non-Native Alaskans, all ultimately completed the program, 19 of whom did so in time to be included in the graduate survey. Thus, the attrition rate was 18.3% for Natives and zero for non-Natives.

Following graduation, 14 of the entering non-Native Alaskans were known to have returned to Alaska where all but 1 were working as physician assistants. An additional 27 non-Native MEDEX graduates were known to have found employment in Alaska. Of this total of 41 non-Native MEDEX graduates working in Alaska in 1991, 28 (68.3%) responded to the graduate survey.

**Professional Background of Entering Alaskans.** The professional backgrounds of the entering Alaskan Native and non-Native trainees differed greatly. As shown in Table 1, the non-Native students came from a

**Table 1.**

### Professional Backgrounds of Alaskan Students Entering MEDEX from 1980 through 1990.

Background	Native	Non-Native	Total
CHA	16*	5	21
Military		1	1
RN		6	6
LPN		1	1
EMT		4	4
Midwife		1	1
Lab/X-ray Tech		2	2
Corrections		1	1
<b>Total</b>	<b>16</b>	<b>21</b>	<b>37</b>

\*p < 0.01

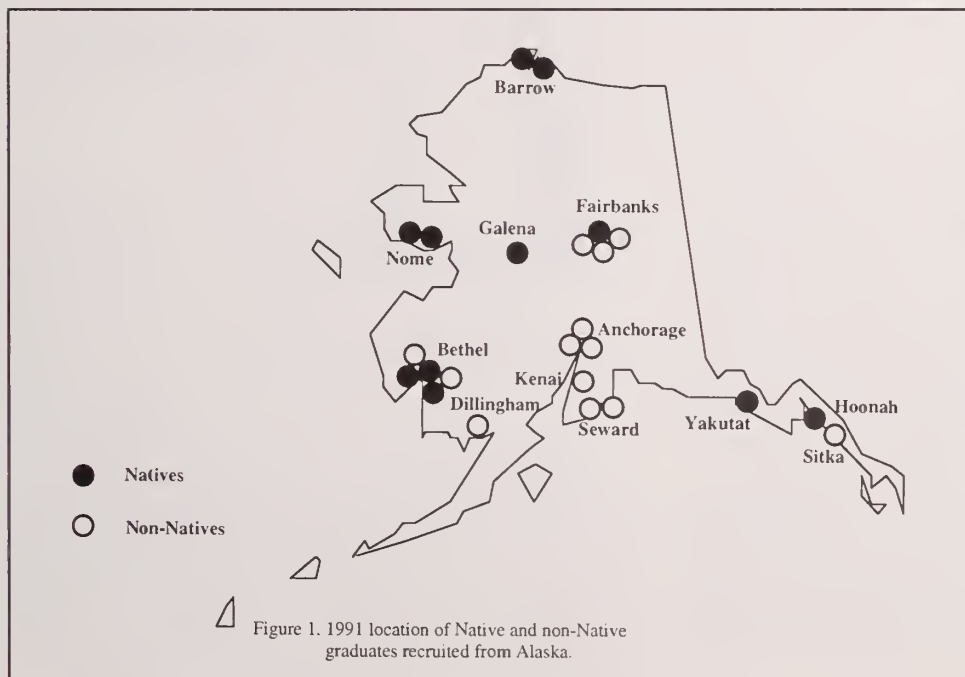
wide range of roles in the health profession, whereas the Native students were all former CHAs.

**Alaskans Returning to Practice.** The Alaskan Natives who attended MEDEX all returned to work in Alaska. Fourteen (67%), of the non-Natives who could be located had returned to Alaska, and 4 (19%) were known to have found work in other states. The remaining 3 (14%) non-Natives either had not yet begun a medical practice or were not located.

It was unusual for Alaskans who completed the program to abandon their careers, and all but one in each

group, 91% of the Natives and 94% of the non-Natives, were still working as physician assistants in 1991.

**Location of MEDEX graduates in Alaska.** Figure 1 shows the geographical distribution in 1991 of Native and non-Native Alaskan students accepted into the MEDEX program. Of the 11 Native graduates, 10 were working in small Native communities of the sub-regional center size (population <2000) and one was working in a Native Health Corporation clinic in Fairbanks



which serves as a referral center for surrounding Native villages and provides care to the urban Native population.

The deployment of non-Native graduates in settings that provide health services to Alaskan Natives was determined both for the cohort of Alaskans accepted into the program and for the total group of non-Native MEDEX graduates working in Alaska in 1991.

In the case of non-Native Alaskans returning to Alaska, six (46%) were working in urban settings either in Fairbanks or Anchorage, and seven (54%) were in rural sub-regional clinics. Of these, two (11% of the total number graduating and 15.3% of those who returned) were working in Bethel, a predominantly Native community, while the other five were in the small non-Native communities of Sitka, Kenai and Seward. For the total group of non-Native MEDEX graduates working in Alaska in 1991, the deployment rate in settings that provide a high percentage of care to Natives was slightly greater, 21%, but still significantly lower ( $p < 0.01$ ) than for Native graduates.

**Demographics of all MEDEX graduates working in Alaska.** The demographic profiles of Native and non-Native graduates working in Alaska were different. As shown in Figure 2 and Table 2, Native Alaskan physician assistants were significantly more likely to be unmarried women in their 30s who grew up in communities with a population of less than 2,500 inhabitants. Non-Native MEDEX graduates, on the other hand, tended to be married men in their 40s who grew up in communities with a population greater than 2,500.

**Practice Environment.** All of the Native graduates and 60% of the non-Natives graduates in Alaska responding to the graduate survey were working in primary care fields. When asked what percent of their time was spent treating patients from different ethnic backgrounds, the Native graduates reported spending an average of 37% of their clinical time treating Caucasian patients and 47% of their time treating Native people, as shown in Figure 3. The non-Native graduates reported spending 23% of their time with Native patients and 61% of their time with Caucasian patients.

When asked to estimate the frequency with which they performed 19 common clinical tasks, the only demonstrable differences between Native and non-Native graduates were significantly more prenatal care and house calls performed by the Natives, as shown in Table 3.

**Table 2.**

**Mean Age and Age Ranges for Native and non-Native MEDEX Graduates Working in Alaska**

	Mean Age	Range
Natives	36.8	32-42
Non-Natives	42	34-58

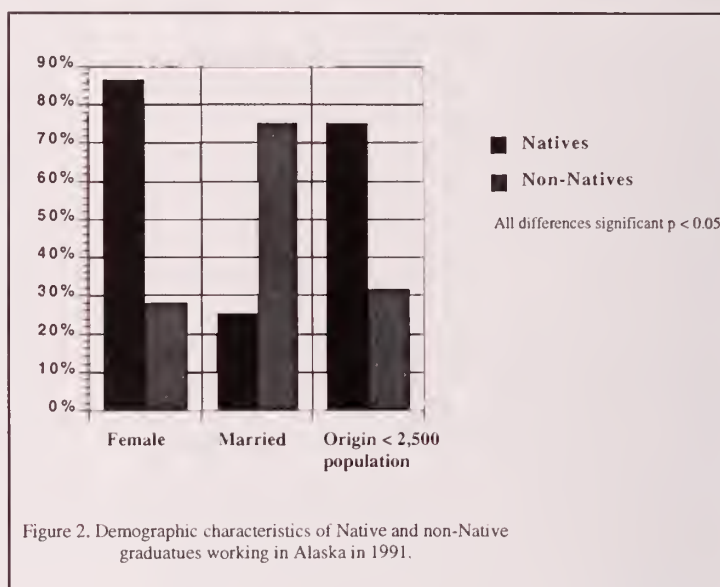


Figure 2. Demographic characteristics of Native and non-Native graduates working in Alaska in 1991.

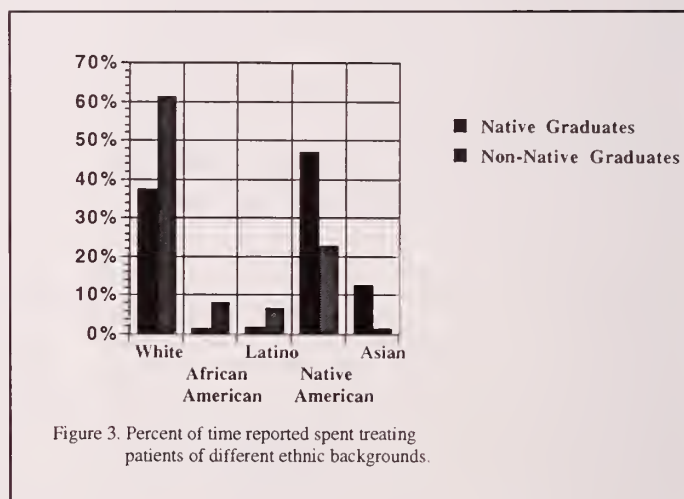


Figure 3. Percent of time reported spent treating patients of different ethnic backgrounds.

**Professional status.** For the graduates working in Alaska there was no difference in the rates of passing the national certification exam for Natives (91%) and non-Natives (100%). The mean salary for non-Natives was higher, as shown in Table 4, although this was largely due to a single outlier. None of the differences in salary or benefits between the two groups was significant.

**Table 3.**

**Number of Times per Month (and range) of  
Clinical Tasks Performed by Alaskan  
MEDEX Graduates**

<u>Task</u>	<u>Natives</u>	<u>Non-Natives</u>
Prenatal care	16 (3-40)*	2.5 (4-40)
Hospital rounds	0.2 (0-1)	4.4 (0-40)
House calls	2.3 (0-8)*	0.9 (0-10)
Nursing home rounds	0	0.4 (0-4)
Supervising	12 (0-20)	10 (0-60)
Discuss pts with MD other than sponsor	12 (0-38)	4 (0-10)
Talk with other PAs	9 (0-30)	6 (0-40)
Emergency work	6 (0-23)	1.6 (0-16)
Casting	4 (0-18)	3.7 (0-45)
Suturing	6.6 (0-18)	6.7 (0-30)
Surgical assist	0	0.4 (0-10)
Delivery	0	0.4 (0-10)
Practice Management	1 (0-6)	5 (1-70)
Personnel Management	0.3 (0-2)	2 (0-25)
Treat AIDS Pts	0	1.5 (0-10)
Hospital committees	0.8 (0-2)	1 (0-6)
Athletic team work	0	1.8 (0-30)
Coroner work	0	0.7 (0-2)
Night call	3.8 (0-10)	2.4 (0-28)

\* p < 0.05

**Table 4.**

**Remuneration and Benefits for Native and  
non-Native Graduates of MEDEX Northwest  
Working in Alaska in 1991.**

	<u>Natives</u>	<u>Non-Natives</u>
Mean Salary	\$39,600	\$51,244
Salary Range	33-48K	24-100K
Vacation Days	13.8 (0-28)	17.27 (0-30)
Sick leave Days	12.5 (0-24)	5.45 (0-21)
CME Days	6.3 (5-10)	6.41 (0-14)

## DISCUSSION

For more than two decades, MEDEX Northwest has been working to develop strategies to maximize the deployment and retention of primary care providers in settings accessible to underserved populations. The most important strategy has been the targeting of specific underserved populations, and training entry level health

workers from those populations to become physician assistants on the assumption that significant numbers of them will return to practice in their communities of origin. Numerous reports have shown that deployment and retention of providers can also be enhanced when clinical training for students takes place in practice sites which serve the target population, thereby providing the students with appropriate clinical role models.<sup>(8)(9)(10)</sup>

Garcia and Fowkes have described a similar approach, selecting physician assistant students from a minority background and training them in clinical settings which served rural and minority communities. They reported deploying 79% of their minority graduates in primary care fields with 60% of them working in Health Professional Shortage Areas.<sup>(11)(12)(13)</sup>

While Native Alaskan MEDEX students left the program at higher rates than non-Natives, attrition was lower than that experienced among minority students in other physician assistant training programs. Weiner and Schneller reported the experience of 48 physician assistant programs in training African-American students for which the overall attrition rate was 65%.<sup>(14)</sup> Our experience with Alaskan Native students points to inadequate personal and family support as the primary cause of failure to complete the program. The lower attrition rates shown here are at least partly due to retention projects funded by the Health Career Opportunities Program featuring a special Bridge Program. This program is designed to assist rural students in the transition to urban academic life, as well as extra tutoring, flexibility in time allowed to complete the program, and strong faculty commitment to training minority students.

The overall outcome of this program can be measured by the fact that the Alaskan Native graduates are working in specialties and in locations that improve the access to care for rural Alaskan Natives. All of the Alaskan Natives went into primary care fields compared with 60% of the non-Native graduates in Alaska. Currently 75% of all MEDEX graduates work in primary care fields<sup>(15)</sup> while in 1991, the national rate for practice in primary care fields for all physician assistant training programs was 53.2%.<sup>(16)</sup> Alaskan Natives are also significantly more likely to work in predominantly Native communities, and they tend to spend more of their time seeing Native patients than do non-Native graduates working in Alaska.

With the exception of a significantly greater role in delivering prenatal care and making home visits, our evaluation was unable to detect any significant differences between the professional responsibilities of Native and non-Native MEDEX graduates currently working in Alaska.



## CONCLUSIONS

We found that the selection of entry level health workers from a target underserved population for physician assistant training, in this case Native Alaskan CHAs, is an effective way to significantly increase the deployment of health care providers serving that population. This strategy requires careful attention to the specific educational needs of the applicants and the cultural factors which make such training a major challenge for the Native CHAs.

## RECOMMENDATIONS

Efforts to improve access to primary care services for underserved minority populations should be based on the principle of selecting students for professional training whose family and personal support systems reside in the target underserved communities. Career ladder opportunities in the health professions for minority entry level health workers should be developed for other urban and rural underserved populations. The outcomes of such training should be evaluated to determine the most effective additional strategies to enhance the deployment and retention of health care professionals in sites providing primary care access to underserved groups.

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# Nursing Staff Educational Preparation and Patient Inflicted Injuries in a 160 Bed Psychiatric Hospital

Roberta Helmuth, RN, BA, MS, CNA, RNC<sup>(1)</sup>

## ABSTRACT

This is a retrospective study of the incidence and severity of patient inflicted injuries upon nursing staff in a 160 bed psychiatric hospital over a period of two years. The investigation explored possible relationships between the basic educational preparation of the injured staff members, the severity of their injuries, and whether they used the behavior management techniques learned in the Mandt System classes. (The Mandt System has been used at Alaska Psychiatric Institute since 1979 to train nursing staff in how to safely deal with assaultive people.)

## BACKGROUND

Acknowledgment of the existence of inpatient violence directed at staff in psychiatric facilities began in the early 1980's. In 1983, Lion & Reid (1) published a compendium of studies about assaults upon staff working in psychiatric institutions. They urged systematic information collection to develop a body of knowledge, as data on inpatient assaults, the number of victims and profiles of the assaulters were largely unknown. In the same year, Soloff (2) identified inpatient violence as a significant occupational hazard for mental health professionals. Lanza (3) found that literature on patient violence was virtually non-existent in the early 1980s. They all reported difficulty in obtaining information about inpatient violence and assault as a result of employee and institutional denial, avoidance, secrecy, excuses and under reporting. Investigation during the 1980s moved from a narrow focus on the frequency of violence and assaults by patients to a more comprehensive study of the subject. The scope of study expanded to include the influence of societal trends, profiles of the victims, the physical and emotional impact upon the victim, risk management considerations, and the effect of training upon the prevention of staff injury. The risk management aspect of working with psychiatric patients has become a significant issue for administrators.

## INTRODUCTION

The Nursing Administration staff at Alaska Psychiatric Institute became concerned early in 1989 when they noted an apparent increase in the frequency and severity of patient care related staff injuries. They asked the Nursing Quality Assurance Committee to investigate the situation, and make recommendations on any problems they identified. The committee attempted to assess both the incidence and severity of staff injuries involving patients to obtain a complete picture of the situation. However, they could find no compilations of this type of data. Nursing Education staff volunteered to design and conduct a research study to collect data to answer the following questions:

1. Has the number of patient assaults on nursing staff increased?
2. Do the levels of medication received by assaultive patients relate to the number of assaults?
3. Has the severity of nursing staff injury increased?
4. Is the Mandt (4) assault management system being used?

The Quality Assurance Committee requested the report of the study findings be routed back to the committee.

Shortly after the study began, the Director of the Division of Mental Health and Developmental Disabilities became interested in the project because he needed data on the possible relationship between medication levels and assaults by patients to present as testimony to citizen and legislative committees working on statutory guidelines for medicating psychiatric patients.

Instrument design, data gathering, analysis, and completion of a focused report on selected study data were accomplished within 6 weeks. That unpublished report (see Summary of Findings—1989 Report at end of this article) provided the Division Director with the information he required. It included the data related to assault frequency, relationship to total hospital census, unit census, patient diagnoses, medication levels and incidence of staff injury.

Tabulation, analysis and reporting of the remaining

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data was not done. Unanswered questions were those related to the severity of staff injuries, educational preparation of staff, and use of the Mandt assaultive management system taught to nursing staff. The following is an analysis and report of that portion of the data.

## METHOD

The population studied was a 164 member nursing staff in a 160 bed psychiatric hospital. The staff composite was 70 Registered Nurses, 3 Licensed Practical Nurses and 91 Psychiatric Nursing Aides. One hundred and forty-four (144) of these positions provided direct patient care. The staff gender ratio was 55% male, 45% female. The study sample comes from a retrospective audit of 1397 incident reports that indicate assault upon staff members. It consists of all reports noting assaults resulting in injury to a staff member. The incident reports cover the period from March 1, 1987, through February 28, 1989.

The categories of data collected by Lehman, Padilla, Clark & Loucks (6), Lion and Reid (2), and Lanza (5) form the basis for the design of the data collection instrument. The questions to be answered were:

- I. Does the basic education level of staff members influence the frequency or severity of patient care related injuries they sustain?
  - A. How many of the injured people were:
    1. Registered Nurses?
    2. Licensed Practical Nurses?
    3. Psychiatric Nursing Assistants?
  - B. How many work days were lost by each of the above groups?
  - C. What was the injury severity as measured by the number of lost work days for each group?
- II. What was the gender distribution of the injured people?
- III. Was the assaultive event training that is required for nursing staff used during this time period?

The audit instrument has 3 sections:

- Section 1. Demographic data about the incident, whether it resulted in staff injury and if so, the name and position code number of the staff person injured.
- Section 2. Information from the assaultive patient's medical records including assault history and medication regimen. (That data was used in the unpublished report mentioned in the introduction of this paper.)
- Section 3. Demographic information about the injured staff member including filing of a Workman's

Compensation form, details of any subsequent claims, and time lost from work. Personnel rosters, Workman's Compensation filing information and reports of responses to calls for assistance were the sources for this data.

Positive responses to the staff injury question in section one identified the study sample and triggered completion of sections two and three of the instrument.

A staff instructor collected the data with the assistance of the medical records supervisor and the personnel officer.

Identification by patient case number protected patient confidentiality. Initially, staff identification by name and position code number enabled data collection from multiple sources. Subsequent conversion of the names to numbers for reporting purposes protected staff confidentiality. The researcher prepared and secured both master code lists.

A "Severity of Injury Scale" developed by this researcher for the study facilitated classification of the injuries. Days lost from work due to injury form the basis for this scale:

- Injuries with 1 work day lost — minor
- Injuries with 2-5 work days lost — minor to moderate
- Injuries with 6-10 work days lost — moderate to severe
- Injuries with over 10 work days lost — severe

## RESULTS

There were 1397 assaults upon nursing staff reported for the 24 month period ending February 28, 1989. Two hundred and sixty-eight (268) staff assault episodes resulted in staff injury. Nine of the reports were incomplete and were rejected. This resulted in a sample of 259 incidents with injury reports.

Findings from the 259 reports:

- Staff member injuries increased from 99 in the first year studied to 124 in the second year.
- Comparison of work days lost as a result of injuries between year one and year two was not meaningful as data for year two was incomplete at the conclusion of the study.
- Some staff were injured more than once - 104 staff were injured in 259 incidents.
- There were 41 assaults with injury that resulted in time lost.
- Assaultive episode injuries resulted in 321 lost work days in the 24 month period. The mean was 7.83 days lost per incident.



- Gender
  - Male nursing staff 72.6% of the injuries  
73.2% of the time lost injuries  
60.1% of the work lost days
  - Female nursing staff 27.4% of the injuries  
26.8% of the time lost injuries  
39% of the work lost days.

Table 1 Gender of Injured Staff		
	Male	Female
259 Total injuries	188 (72.6%)	71 (27.4%)
41 Time lost injuries	30 (73.2%)	11 (26.8%)
321 Work days lost	193 (60.1%)	128 (39%)

Review of the basic educational preparation of the injured staff showed Registered Nurses reported 61 injuries; 9 involved lost time, for a total of 124 days. One Licensed Practical Nurse reported an injury, with 1 day lost. Psychiatric Nursing Aides reported 197 injuries; 31 involved time loss, with 195 days lost.

Table 2 Injuries by Position Categories			
Total #	Registered Nurses	Licensed Practical Nurses	Psychiatric Nursing Aides
Injury Reports	61 (23.6%)	1 (.3%)	197 (76.1%)
Time Lost	9 (22%)	1 (2.4%)	31 (75.6%)
Injuries			
Days Lost	125 (39%)	1 (.3%)	195 (60.7%)

Severity as measured by time lost, showed 10 occurrences in the 1 day lost category, 20 in the 2-5 day category, 3 in the 6-10 day category and 8 in the above 10 day category.

Table 3 Severity as Measured by Time Lost		
1 day lost (minor)	10 occurrences	24.4%
2-5 days lost (minor-moderate)	20 occurrences	48.8%
6-10 days lost (moderate-severe)	3 occurrences	7.3%
10+ days lost (severe)	8 occurrences	19.5%

Four of the injured staff members had not participated in Mandt training. The remaining 98.5% of the staff reporting injury had participated in the training. Respondents in 98.1% of the incidents reported using the Mandt system techniques.

## DISCUSSION

The data collected in this study are consistent with the studies of Hodgkinson, Melvor & Phillips (7), Lanza (3 & 5), and Blair (8). They reported the staff with the least education sustain the greatest number of injuries.

Our investigation found the injury rate for the paraprofessional group, the Psychiatric Nursing Assistants, was 3 times higher than that for the Registered Nurses (76.1% compared to 23.6%). These figures closely parallel the 72.6% male to the 27.4% female injury ratio. The facts that 55% of the staff are Psychiatric Nursing Assistants (predominately male) and 45% are Registered Nurses (predominately female) may explain some of the difference; it does not explain the entire disparity. In previous studies, gender reporting is either missing or not meaningful.

Blair (8) postulated that staff with less basic education (the paraprofessional groups and students) tend to be less skilled in using effective communication techniques. He observed these staff members have a tendency to rely more upon authoritarian communication styles that may tend to provoke some of the assaultive behaviors. Studies have found over-controlling environments provoke increased anger and violence from the groups being controlled.

The impact of more intensive communication skills training upon incidence of assaults needs study. The effects of training staff to allow the patients more autonomy and freedom of choice also needs evaluation.

Psychiatric Nursing Assistants have the longest periods of direct contact with the patients. They are some of the first people to respond to emergency assistance calls and have the initial contacts with the person who is out of control. This places them in a higher risk situation and may have an impact on the injury rates. Rice, Harris, Varney and Quinsey (9) also pointed out this factor.

Few studies focused on the severity of injury. There was no agreement on how to measure severity of injury. Consequently, there was no quantification of study results. There was no development in previous studies of uniform criteria for the assessment and measurement of severity of injury that could provide quantified baseline data for comparison. Hunter & Carmel (10) measured the severity of assaults by calculating the cost to the

employer. They felt staff assault is a significant risk management issue and advocated an accurate loss analysis be done to measure the total cost of employee injuries from patient violence. Nigrosh (11) measured severity by calculating an average of the number of days lost per employee per year.

The Severity of Injury Scale developed by this investigator for this study measured severity by the amount of time lost from work (Table 2). The data indicated the Psychiatric Nursing Assistants lost the highest percentage (60.7%) of time. Approximately 25% of the lost time injuries fell in the mild category, almost 50% in the mild to moderate, and the remaining 27% in the moderate to severe and severe ranges. (This study does not include variables relating to job experience and staff-to-patient ratios.)

Lion (2) advocated mandatory training for direct care psychiatric staff, psychiatrists and physicians who work with assaultive patients. Infantino and Musingo (12) found that staff trained in aggression management techniques suffer fewer injuries.

Data from this study indicated 98.5% of the injured staff had participated in the assaultive behavior management training (Mandt system) provided by the hospital. It does not appear that failure to use the system contributed to the injuries since more than 98% of the assaulted staff had received training.

Several studies analyzed assaultive episodes and found that containment and restraint situations result in more serious staff injuries than battery situations. A "containment and restraint situation" is one in which a combative patient is forcefully subdued, and a "battery situation" is one in which an unprovoked and unexpected attack occurs by a previously noncombative patient. Rice, et al (9) separated staff injuries into those two categories and modified their procedures and training as a result. Hunter & Carmel (10) also differentiated between battery and containment injuries to staff. Differentiation of battery injuries from containment injuries could form the basis for additional study at Alaska Psychiatric Institute. Modification of the Mandt System techniques merits consideration if the findings indicate that a greater number of injuries occur during containment than from direct assault.

Another variable mentioned in the literature, but not addressed in this study is employee morale. Poor morale may have an impact on the amount of time an injured person remains off work following an injury. Rice, et al (9) proposed that the injured employee who does not feel good about the work situation may measure and/or report a severity of injury greater than sustained. That person may then remain off work longer than may be medically necessary, resulting in an inflation of the severity figures. (They did not mention whether they

included the use of time for psychological healing, as well as physical healing.) They postulated that if the time lost from work factor correlates with prior use of sick time by the injured individuals, it could be a predictor of risk for injury for other staff. They proposed consideration of staff morale as a variable in future studies.

Efforts to study inpatient violence started in the early 1980s with the acknowledgment that it existed. Early researchers continued by examining the frequency and only recently have studied the severity of the attack and the resulting injuries. Factors such as the milieu, administrative support and guidance, staff gender and basic educational preparation of the staff have received very little attention. Lack of comparability across studies remains a problem. Risk management research is only beginning to investigate how injuries occur and how to reduce the incidence of injury. There is no standard definition of assault. There is no theory of inpatient violence. There is no general acknowledgment of the moral and ethical responsibility of the hospital's administration to both staff and patients. Attitudes about violence as a common, expected and accepted occurrence in inpatient settings need examination. The influence that acceptance of violence in society has upon violence in hospitals is unknown.

If there is as much progress in this decade as there was in the 1980s we may find an answer to some of the questions about inpatient violence. However, there is general agreement that serious injury of staff members is less likely if they practice prevention and safe management of violent episodes.

## 1989 REPORT SUMMARY OF FINDINGS

There were 1397 assaults on staff members reported over a 2-year period. Two hundred fifty-nine (259) of them resulted in injury to the person assaulted.

Significant findings about the assaulters were:

- The primary diagnosis of 70.1% was schizophrenic disorders, other psychotic disorders or mood disorders.
- Forty-seven percent (47%) were either refusing to take medication or had no medication ordered.
- More than half (54%) had a history of assaultive behavior.
- A small number of patients (3) were responsible for 19.7% of the injuries.

No correlation was found between the number of assaults with injury and total hospital census, individual unit census or type of unit.

*(continued on page 203)*



# Letters to the Editor

Dear Editor

I have been the medical director of the Iliuliuk Clinic for about seven years. For those of you not familiar with this clinic, it is located approximately 800 miles "on the chain" at Dutch Harbor-Unalaska. This is a remote "bush" clinic dealing with a local population, as well as the fishing industry. A lot of minor and major trauma and medical cases present to this clinic. These cases require triage, stabilization, treatment, a return to work, or transportation off "the rock" for additional treatment or convalescence.

The clinic is staffed by physician assistants and nurse practitioners 12 months a year and physician specialties care for about eight months of the year. The specialty care is scheduled based on need; i.e., orthopedics during the fishing season, pediatrics during spring and fall, OB-GYN, emergency care and family practice all quarterly.

In early 1994 I had the opportunity to allow a 4th-year medical student to spend one month working in the clinic with each of us. This student doctor, Ellen Kim, so impressed me with her desire to learn and teach at the clinic, I reconsidered my previous stand against student physicians. During her stay on "the rock" at Dutch Harbor-Unalaska we all learned to respect her and truly enjoy her company.

The physician assistant, Jim Bird, asked her to write a brief summary of her impressions of our clinic and her visit. The following are her impressions.

Donald G. Hudson, D.O.

This was not my idea. Jim, the physician assistant, asked me to write about my impressions of Dutch Harbor. I am a 4th-year medical student from New Mexico doing a one-month rotation at the clinic. My friend of many years, Siobhan, is here for the fishing season, working as a vessel agent of sorts, and it is because of her that I found myself here. I love this place of contrasts and extremes where life seems simpler and paradoxically, more challenging. It keeps me awake at two o'clock in the morning when I have been at the clinic for fourteen hours, and then go down to city dock to find Siobhan reconciling the off-load tally. Another vessel agent whom I met in the clinic recognizes me and offers us a tour of the Korean tramper which smells of ginseng. . . .

The day I landed the wind was enough to make the enormous man seated next to me talk of death. He came to work on a crab boat, a livelihood which is notoriously dangerous. Could landing on this airstrip with water on either end be as hazardous as crabbing? Even the Mark

Air flight had a taste of adventure to it. The first day's fear of landing was inconsequential next to the second day's fear of my incompetence, however. I had almost no trauma or clinic experience, having done most of my rotations with hospitalized patients. Even more intimidating, everyone at the clinic knows not only their job, but something of everyone else's. For example, I met the radiology technician in x-ray only to meet her later in the day as the lab tech/phlebotomist, only to meet her again after hours as a volunteer Emergency Medical Technician. Such is the life of a health care provider in a more remote geography. I liken myself to a mental sponge, absorbing as much as possible, seeing things I may never have the opportunity to see again. There is no pharmacist so I fill my own prescriptions. I have learned to make copies of x-rays, (although I haven't shot any films, not wanting to irradiate anyone excessively). I watch the lab people do lab things and I am grateful that I don't have to. The visiting orthopedist demonstrates his musculoskeletal expertise in the treatment of the generous number of occupationally incurred accidents/injuries: avulsions, lacerations, fractures, burns, sprains and spasms. Almost everyone has carpal tunnel syndrome and/or tenosynovitis. I think I may be developing carpal tunnel in sympathy for them. It's amazing how many patients come in with the same story, "a wave hit the boat and ten fifty-pound boxes fell on my \_\_\_\_\_," (fill in the blank with any body part) or "I'd been working eighteen hours when all of a sudden my hand was caught in the conveyer belt." Many charts carry the diagnosis of "crush injury." Sounds bad, doesn't it?

This past week I have been working with the physician assistants and nurse practitioner, seeing odd and wondrous things, again, mostly occupational injuries such as hernias, crab asthma, and skin rashes extraordinaire. I have now seen my first patient with trench foot, something I had only read about in Ernest Hemingway novels. I treated someone with scabies the other day, and even as I write this my skin is itching unnervingly. Tinea versicolor. This sounds more like something fragile, foreign, and perhaps kaleidoscopic than a body fungus. And did you know that you could get trichinosis from walrus meat or that the bite of a seal is potentially much more dangerous than that of a pit bull?

After obtaining the history and physical exam from a patient, I present this information to the PA or NP who then sees the patient. We discuss the best treatment options, which are often limited by the fact that the patient will be out at sea again tomorrow. The PAs and NP here have more experience than most physicians I  
(continued on page 204)



# Sexually Speaking. . .

Mary B. Cavalier, M.S.

## 'TIS THE SEASON . . . FOR ANTIDEPRESSANTS

Winter is upon us with all of its sparkle, joy, excitement and for many, dread. This is the season where many people seek relief from the darkness, holiday blues or cabin fever by using antidepressants. The one possible side effect of many of the popular antidepressants such as fluoxetine (Prozac®) and sertraline (Zoloft®) is a pharmacologically induced sexual dysfunction.

Although a decrease in sexual arousal and/or desire may be a symptom of depression and may be relieved with an antidepressant, pharmacologically induced sexual dysfunction manifests itself after the beginning of treatment. Common symptoms include:

- Loss or reduced sexual desire
- Inability or reduced ability to become sexually aroused during sexual activity
- Erectile disorders
- Premature ejaculation
- Retrograde ejaculation
- Painful erection
- Inability to achieve orgasm
- Change in lubrication
- Pain during sexual intercourse

Yohimbine (Yocom®) has been demonstrated to be effective to combat the side effect of erectile dysfunction in men. It does not address the side effect of reduced desire. Also, whether it is effective in women is not known due to the subjective quality of sexual satisfaction in women.

Two antidepressants do not inhibit sexual function. Those are trazodone (Desyrel®) and bupropion (Wellbutrin®). Since the later is taken three times a day, it does have a lower patient compliance rate and also the side effects may not be tolerable to some patients.

It has been my experience that patients who have been fully educated about the potential side effects choose to maintain treatment with antidepressants. On the other hand, patients who were not aware of the side effect of sexual dysfunction often personalize the lack of functioning and therefore, add to the depression and hopelessness. Therefore, patients should be fully educated on the potential side effects of antidepressant on their sex life.

As the saying goes, a well informed patient is a happy patient!

In closing, I hope the New Year brings all of you wonderful surprises!

*A special thanks to Robert Alberts, MD for consulting with me on this article.*

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## Clinical research: Women must be included

The AMA believes medical research needs to involve women in all clinical trials that can have an impact on women's health.

AMA Trustee Palma E. Formica, MD, delivered this AMA message to the Office of Research on Women's Health. She discussed issues and strategies for increasing the recruitment and retention of women in clinical studies.

Dr. Formica also recommended increasing the number of female biomedical researchers and the number of female researchers in leadership roles and other positions of authority.

"Inconsistencies exist, often to the disadvantage of women's health," Dr. Formica noted. "Gender-related factors are ignored frequently when health interventions derived from studies of men are generalized to the entire population."

She said that additional costs to include women in clinical studies are vital to "learn with confidence whether findings do indeed apply to the population at large." Women respond differently to treatment than men.

# History of Medicine in Alaska

EDWARD D. SPENCER, M.D.

On the 13th of June, 1920, Edward D. Spencer was delivered by Dr. West on the couch in the dining room of the family home in Woodstock, Illinois. Ed. was the thirty-third baby born in the county after the collection of statistics began.

His father followed a trade called "japanning," which is a technique for painting the insides of typewriters, the manufacture of which was the chief industry of Woodstock. During the Depression, Mr. Spencer's time was gradually cut back until the plant closed. Ed's oldest sister, who was 24 years older, helped support the family. Later, she helped him with the first year's tuition at medical school.

He remembers the joy of playing baseball in the summer. He walked a mile to high school, where he had straight A's and was rewarded with a watch, which he promptly lost.

He helped his family financially with various jobs which meld in memory. He delivered for a meat market, worked on an ice truck carrying 100 lbs. of ice up backstairs and handing out ice chips to the kids, and had a job in a water treatment plant. He managed to save \$240 for tuition at college.

He became a deeply convinced Christian which led him to Greenville College in Greenville, Illinois. He was granted a work scholarship to make up the difference between the money saved and the \$400 needed for tuition, room and board. During the war, he was in college studying chemistry. His mother was now a widow and their family physician Dr. Sandeen, was chairman of the draft board. He told Ed Spencer that "we don't take chemists" so he was deferred until after graduation.

When on May 31, 1944, he was called up, Ed was rejected by the Army for a medical disability. However, he had applied for a commission in the Navy but his medical condition also precluded naval service. He regrets not being able to go to Princeton University for naval indoctrination.

He tried to take an advance course in chemistry at the University of Chicago primarily to teach at Greenville College. He believes he was interviewed for this by one of the atomic scientists who dissuaded him from taking the course. However, he taught chemistry for one year and eventually turned down a job at Oak Ridge working on the atom bomb.

While he tested high for medicine on an aptitude test, he was torn between it and the ministry. Finally he chose medicine.

Once in medical school he did the best thing in his life aside from committing his life to the Lord. He married Mary Short, an RN from Johns Hopkins Nursing School,

who was teaching at the Presbyterian Hospital. She was able to see him through medical school. During the next three years they lived in an apartment near the medical center.

The Spencers went to Seattle to King County Hospital, Harborview for his internship. While on the one hand, the social workers screened patients to see if they could pay; on the other, it had the only emergency room in the area. Dr. Spencer remembers a patient who was asked why, if she could afford to maintain an apartment, she couldn't pay the hospital. The patient sat up on the stretcher indignantly. As he recalls, she went elsewhere. He remembers the young girl who came in DOA with diphtheria. When he examined her, he identified the white membrane in her throat. Her doctor was in the operating room, scrubbed but he could not have helped her. Dr. Spencer learned a great deal in this busy place with its variety of problems.

He was commissioned in the Public Health Service and sent to Sitka, Alaska in 1952. He was attached to Mt. Edgecumbe Hospital which was operated under the Department of Interior as the Alaska Native Service Branch of Health which by contract employed doctors, some were conscientious objectors; others PHS, and still others civil servants.

Both of the Spencer sons were born in Sitka. Mark, in 1953, now practices in Arlington, Washington and John, in 1954, who practiced in Anchorage, but is now on a fellowship at the University of Tennessee Medical School.

After living in Alaska, the Spencers went to the University of Colorado Hospital for a Residency in General Practice, now Family Practice, which was one of the first in this field in the country.

He took a locum tenens in Wessington Springs, South Dakota for a doctor serving in the military. Both of the Spencer sons were incubating measles and on arriving in Wessington Springs engendered a small epidemic which, as Dr. Spencer wryly observes, provided him with a busy practice since he brought his own disease. The other doctor in town was so tired that one day he climbed in an empty bed in the ward and went to sleep.

However, Sitka seemed the place to practice. They rented a house and the parlor became the waiting room, the back bedroom the office-examining room, the bathroom the lab. Mary Spencer served as nurse-receptionist as well as wife-mother. In addition, she was on the library board during the time the new library was being built. After six months, they moved the



office to a building built by the Russians which was slowly sinking into the sea.

For thirty-three years Dr. Spencer was medical officer for the Pioneer Home which was the only one in the state for much of this time.

When the pulp mill was constructed, Sitka became loud especially at night. He treated one worker who had been overcome by accidentally released gas. Another, who while tipsy, cut his head. He was duly stitched up and billed. The bill was returned with the questions: "Dr. Spencer, who are you? I don't remember you. Where did I meet you?" And the bill was not paid.

One night he received a call that a lady at the Alaska Hotel wanted him. Apparently, the manager of the telephone system monitored calls and Dr. Spencer was told to pay no heed to the request. Some days later, while walking the beach, a prominent woman in Sitka found a thumb. Soon the body parts were assembled. It transpired that the lady at the Alaska Hotel, after a beating by her husband, killed and dismembered him. Another doctor, Isaace Knoll, had answered the call and treated the victim before he died. Dr. Knoll testified at the subsequent trial and then left for Cleveland.

Sitka acquired a mortician in 1959 who was perturbed by a body found floating in the bay with a pencil sized hole in the chest over the heart. He called the

coroner who found it "death by drowning and added 'probably caught on a nail under the dock.'"

A patient gave Dr. Spencer her father's ledger, written in Eagle from 1911 to 1918. The father claimed to be a physician but Dr. Spencer believes he was probably a pharmacist or herbalist who, like barbers, were prominent in the history of medicine. The ledger belonged to Dr. Fox from England. It contained prescriptions for chilblains, whooping cough, gonorrhea, etc. Dr. Spencer has been tempted to send one of these to a pharmacist to see if it could be filled today when druggists dispense from shelf bottles rather than compound the prescription. Also there was a clinical section in which the author describes scanty water, fever and then added VT - visit and treatment - \$5. There was a patient who was sent to the hospital in Dawson but who died en route. It was necessary for Dr. Fox to sign the insurance papers attesting to the death which, of course, had to do with payment of life insurance. This ledger has been given to Elva Scott of Eagle who established the Eagle Historical Society.

Through the years, he befriended several physicians. Dr. Philip H. Moore, an orthopedic surgeon, had been authorized to set up a clinic on the deactivated Naval Base on Japonski Island which evolved into Mt. Edgecumbe Hospital. This was a 350 bed hospital, 50 of which were a pediatric orthopedic ward. Dr. Moore pioneered freezing bone for spinal fusions, using the bone from the thorocoplasty surgery for tuberculosis.

Dr. Moore went into private practice in Sitka and became actively involved in the community, as well. He was instrumental in the establishment of the Sitka Community Hospital in 1956. Also, he persuaded the Chamber of Commerce to establish a health plan with Blue Cross. This was so successful that Blue Cross lost money.

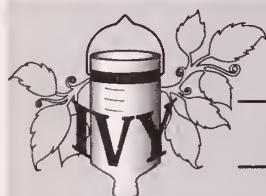
When Dr. Moore retired, Dr. George Longenbaugh came, a good surgeon with excellent judgement. When a man almost bled to death as a result of a logging accident, Dr. Longenbaugh devised a first aid program for the men in the logging camps.

Dr. Edward Spencer had always wanted to be a missionary and it was not surprising that he felt "the Lord wants me further north." Consequently, when he was asked to be medical officer at the Palmer Pioneer Home, he accepted and moved north.

This past spring, Ed Spencer went back to his 50th college reunion. He thoroughly enjoyed it. Especially when he went into the bookstore realizing that he had never gone in it before with money in his pocket.

He says simply that he could not have done it without his wife. Dr. Edward Spencer comes forth as a committed, modest man who relates warmly to others.

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# ALASKA MEDICINE

Volume 6, Number 2

June, 1964

EDITOR'S CONVENTION COPY



# From Out of the Past . . . Over Thirty Years Ago

Gloria K. Park, M.D.

*Providence Hospital received the majority of quake injuries as initial reports stated that the Alaska Native hospital was destroyed and that the Presbyterian and Elmendorf hospitals were being evacuated. Following are quotes from the "Quake issue" of Alaska Medicine:*

**From Elizabeth A. Tower, M.D. (Anchorage) —**

"We were almost unbelievably luck both in the paucity of casualties requiring treatment and in the miraculous sparing of the medical facilities. As the pictures in this issue will illustrate, very slight changes in the configuration of the slide areas might have placed the Alaska Native Service Hospital in a position similar to that of the Government Hill School—split in two with one half at the bottom of a 50 foot cliff—or might have rendered the Presbyterian Hospital building or St. Mary's Residence a mass of rubble like the four Seasons Apartment across the street. A half block shift in the L Street Slide would have wiped out not only two hospitals but the offices of half of the physicians in Anchorage."

**From Walter Johnson, M.D. (U.S.P.H.S. Hospital - Anchorage) —**

"Although the bluff behind the Alaska Native Hospital yielded to the quake and gave way in places, the building stood. Patients on the fifth floor found their beds tossed across the room but no patient was seriously injured. They took this, as they have many other disasters, with their usual equanimity. Thousands of medical charts and x-ray folders were shaken from their files and the file cabinets turned over on them. The x-ray panels were wrenched from the walls. Hundreds of bottles of drugs in the pharmacy danced off their shelves and smashed on the floor. In the storeroom stacks of cases of food and supplies were toppled. The sprawling building was erected as four distinct units. During the quake each wing rolled separately, the building dividing neatly at the 'crumble joints' as it was designed to do. Without these the building would certainly have been torn apart in a haphazard manner. Two steam pipes were sheared off where they passed through the main beams at the 'quake joint'. Several days were required to repair this and other breaks in the steam pipes."

**From J. B. Deisher, M.D. (Seward) —**

"After a devastating earthquake, fire, and tidal wave, which removed the shoreline of the city of Seward, the medical problems were astonishingly small and largely related to Public Health and sanitation. The minimal number of actual injuries related to this disaster allowed

the citizens of the community to continue to manage the medical problems without help from outside other than the importation of a sanitarian, a pharmacist, a Public Health laboratory technician, and a relief Public Health Nurse, after the first week."

**From J. Bruce Keers, M.D. (Kodiak) —**

"The Griffin Memorial Hospital suffered no quake or water damage, although an after shock at 12:55 p.m., April 14 (6+ or so on the Richter scale) caused a few plaster cracks. Our solid rock underpinning here in Kodiak probably saved us from severe structural damage during the quake.

The hospital lost electrical power when the first wave took out the cooling system running out into the channel from the power plant at KEA. With darkness descending upon us and the terrible commotion of rushing water, crashing and grinding noises, etc., just outside the hospital in the channel as all the many docks, canneries, and other facilities were being swept away, and finally because the water was becoming covered with oil and gasoline from the severed Standard Oil Company lines, it was decided to evacuate the hospital. Of some eight or nine patients present at the time, a few were allowed to leave to stay with friends or relatives in their homes. The other half dozen were taken up to the high school and bedded down in one of the rooms in charge of three of our nurses. The high school, incidentally, became the major evacuation point for all people from the low lying areas, and remained so for the following week.

One humorous incident: when the power scow *Selief* contacted ACS just after the third and highest wave, and was asked for his position, he replied that he was on the side of the hill directly down from the downtown school."

**From Ed Fortier (Providence Hospital, Anchorage) —**

"The hospital was operational with these deficiencies:

- a. For an unknown period of time, essential power would have to be provided by the 125 KW auxiliary diesel plant. Chief Engineer Philip McLean advised the auxiliary power unit could operate at least 72 hours without difficulty.
- b. Internal communications were out, with the exception of the broadcast or paging system.
- c. External communications were totally inoperative.
- d. Water mains had been broken and additional water would be required to maintain the boilers as well as operate toilets.

(continued on page 202)





*U. S. Army Mohawk Photo*

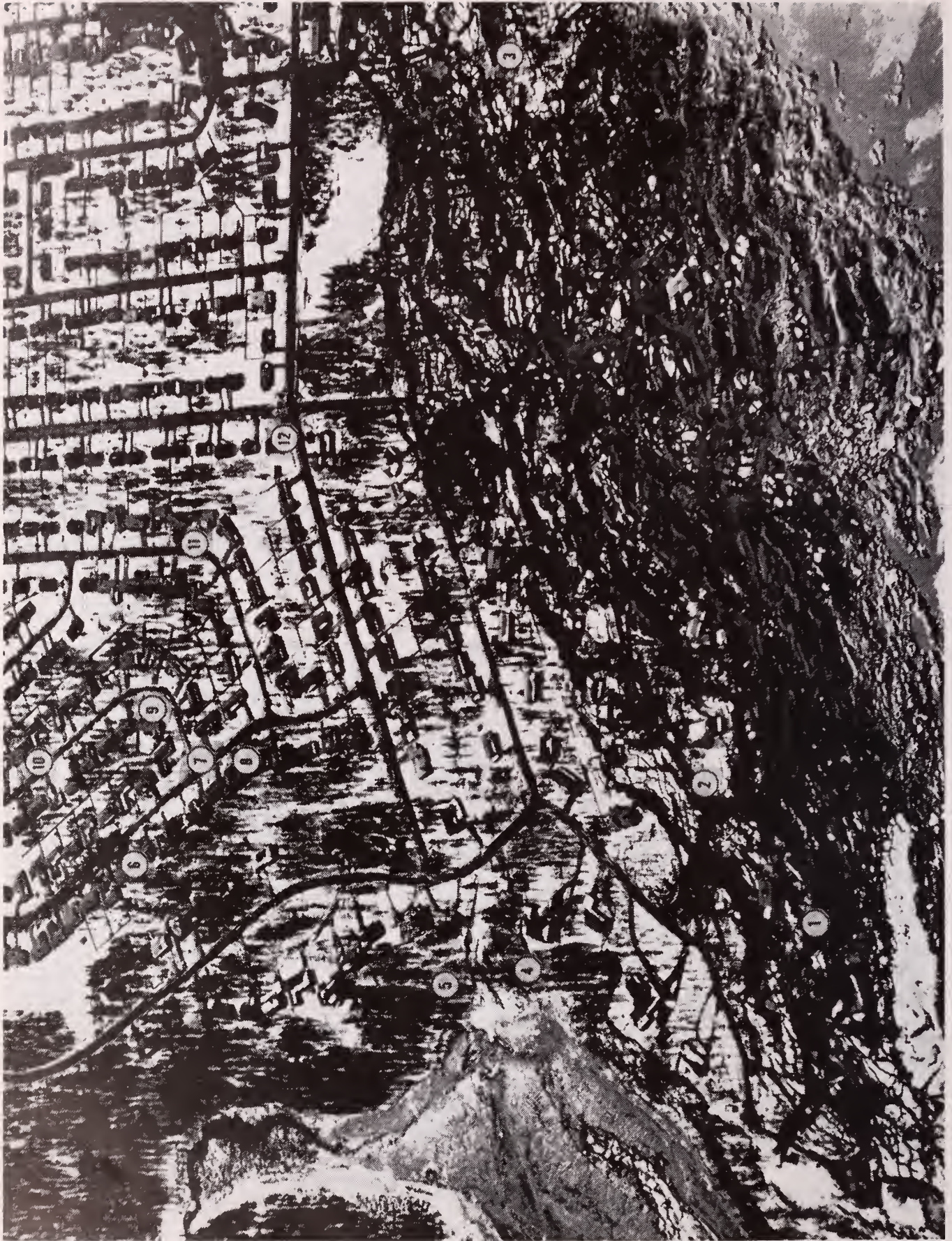
Alaska Native Hospital at Anchorage and First Avenue Slide following March 27, 1964 earthquake.



*United States Air Force Photo*

A typical wall of USAF Hospital Elmendorf following the Good Friday Earthquake. Total damage is estimated at approximately \$2,600,000. Evacuation of all patients was accomplished in eighteen minutes and a temporary 200-bed hospital utilizing nearby barracks was made operational in thirty-five minutes. Normal hospital operations were reestablished on 6 April 1964.









**TELEQUANA BLUFF AREA, ANCHORAGE**

(Above)

*Mac's Foto*

1. Area of the residence of Dr. Perry Mead near which his sons Perry, 12 and Merrill, 2 were last seen.
2. Residence of Dr. Richard Sutherland
3. Residence of Drs. Theodore and Rosalie Shohl
4. Lot upon which the Drs. Shohl planned to build in the summer

**TURNAGAIN BLUFF AREA, ANCHORAGE**

(Facing)

1. Residence of Dr. Asa Martin
2. Residence of Drs. Robert and Helen Whaley
3. General area of the residence of Dr. William Caughran
4. Residence of Dr. Jack Sedwick
5. Residence of Dr. Francis Phillips
6. Residence of Dr. Robert Wilkins
7. Residence of Dr. Charles St. John
8. Residence of Dr. Royce Morgan
9. Residence of Dr. J. Ray Langdon
10. Residence of Dr. Mahlon Shoff
11. Residence of Dr. Don Val Langston
12. Residence of Dr. John Pennington

*Mac's Foto*



*(continued from page 198)*

- e. The Radiology department was without power for operation of major x-ray units.
- f. The laboratory and pathological area was without power for operation of equipment or lights.
- g. Extensive breakage had occurred in the pharmacy area.
- h. The kitchen area was without power and in serious disarray.
- i. The elevators were inoperative, although by 6 p.m., the engineers had determined that no persons were trapped in them.
- j. The exhaust fans within the hospital were not functioning.

To my best knowledge, there is no absolutely

accurate figure on the number of persons who received medical treatment at Providence Hospital during the first 48 hours after the earthquake. An initial survey indicated 108 persons passed through the Emergency Room treatment area from 6 p.m. Friday to noon on Saturday, with eight casualties admitted. During the period from Saturday noon, March 28, to midnight on Sunday, March 29, the initial report indicated an additional 89 persons were recorded as being processed through the emergency room with a total of seven admitted as casualties. In addition to these totals, 22 patients from Presbyterian Hospital were evacuated into Providence, and an additional 27 from St. Mary Residence were moved into the hospital in the first 48 hours.



Power scow *Selief*, "on the side of the hill directly down from the downtown school"



(continued from page 192 — Nursing Staff)

The study findings were consistent with those of other researchers.

## ACKNOWLEDGMENTS

Appreciation to: Heather McCracken, RN for data collection and collaboration; Reta Sullivan, Medical Records Supervisor for the assistance provided in accessing incident reports and medical records; and Jim Gordon, Personnel for the data on Workman's Compensation claims.

*Submitted with permission from the Director of Alaska Psychiatric Institute.*

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**Action:** Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage, although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalamic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it, however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

**Indications:** Yocon<sup>®</sup> is indicated as a sympatholytic and mydriatic. It may have activity as an aphrodisiac.

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**Adverse Reactions:** Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.<sup>1,2</sup> Also dizziness, headache, skin flushing reported when used orally.<sup>1,3</sup>

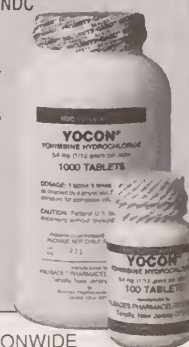
**Dosage and Administration:** Experimental dosage reported in treatment of erectile impotence.<sup>1,3,4</sup> 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to 1/2 tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.<sup>3</sup>

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*(continued from page 193—Letter to the Editor)*

have ever worked with and it shows in the teaching they share with me, whether it concerns the treatment of hypercholesterolemia or how to build a cabin. They have also taught me how to use their audiometric, tympanometric, and spirometric devices. The nurses are just as wonderful, supervising my first IVs, electrocardiograms and intramuscular shots. When the staff is not sharing their stories with me (of ships and shoes and ear wax, and the span of eagles' wings...), they are sharing their halibut, which is just as appreciated.

I have assisted in minor surgeries on carbuncles of the buttock, lectured a junior high class on cardiovascular physiology, heard men from Ethiopia, Russia, the Philippines, Korea, Mexico, Vietnam, Czechoslovakia and Montana swear in their native tongues. I have heard of the stoicism of these people who work on boats. One Japanese captain lines up his workers at the end of every day for a hand inspection to check for broken fingers and partial amputations. Otherwise, the injured would continue to work without any medical attention. I have seen these same men laugh uncontrollably at their ship mates when they see the Teenage Mutant Ninja Turtle band-aids I have placed on their wounds.

After a day at the clinic, in awe of those who work here and the patients I have seen, I encounter the same people at the grocery store or Stormy's restaurant. I love this place. Where else can you see an eagle and seal fight for a fish or the surreal view to the west whilst careening face first down Pyramid's icy incline? When I return to New Mexico I will try to describe Dutch Harbor: the mud, the steam from Makushin on a clear day, the smells of fish, of a smoke filled ship's galley between shifts, of the antiseptic clinic, of wet dogs, propane and the interior of a battered four wheel drive impregnated with the scent of sweaty work clothes and overripe produce, the sounds of someone whistling "Strangers in the Night" while on call in the emergency room, tires on slushy gravel roads, wind challenging the winter-worn wood of the cabin at Ski Bowl, radio squawks between ship and shore, ravens and sea lions and the silence of frozen, tundra-blanketed mountains.

Once again, it is two o'clock in the morning, and I am wide awake.

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# GLIMPSES OF ALASKAN MEDICAL HISTORY

Edited by Robert Fortune, M.D.

## THE JOYS OF A RUSSIAN STEAM BATH IN NEW ARCHANGEL (1841)

Sir George Simpson (1792?-1860) was born in Scotland and received his schooling in London, where in 1820 he joined a mercantile house. He was soon posted to New York and then to Montreal with the Hudson's Bay Company, which placed him in charge of the Athabasca fur-trading district. In 1821 the North West Fur Trading Company merged with the Hudson's Bay Company and the young Simpson, who had already shown great promise for his administrative ability, was promoted to Governor of the Northern District, known as Rupert's Land—a vast tract encompassing the greater part of present-day northwestern Canada. He later became the administrative director, or Chief Factor, of the entire operations of the Hudson's Bay Company in North America. He traveled constantly by canoe, sledge, and snowshoes throughout his "realm," visiting distant fur-trading posts, sometimes unexpectedly, and all the while keeping a lively and detailed diary. He also warmly encouraged arctic explorations by his fur-traders, one of the most significant being that of his nephew Thomas Simpson, together with Peter Dease, who descended the Mackenzie River by boat and passed westward along the arctic coast to Barrow in 1837. Over a decade later he also encouraged the important journeys of Dr. John Rae, a fur-trader/physician who was instrumental in solving the mystery of the disappearance of Sir John Franklin in northern Canada.

In 1841, Queen Victoria bestowed a knighthood upon him in recognition of his exceptional services on behalf of the empire. That same year he set off on a journey around the world, the greater part of it overland, during which he crossed Siberia from the eastward all the way to St. Petersburg. In the course of his travels he visited Sitka, in September 1841, where he was received by his Russian counterpart in the Russian-American Company, Governor Adolph Etholen.

Simpson's impressions of Sitka were distinctly unfavorable, if we may judge from his diary entry in March 1842, when he briefly returned: "Of all the dirty and wretched places that I have ever seen, Sitka is pre-eminently the most wretched and most dirty. The common houses are nothing but wooden hovels, huddled together, without order or design, in nasty alleys, the hot-beds of such odors as are themselves sufficient, independently of any other cause, to breed all sorts of fevers. In a word, while the inhabitants do all that they can to poison the atmosphere, the place itself appears to

have been planned for the express purpose of checking ventilation."

During his stay in the Russian capital in 1841, Simpson spent a few days ashore but retreated to his ship's cabin for the nights, perhaps because of the noisome environment he describes. Ever alert for new experiences, however, he apparently couldn't resist the opportunity to try a steam bath of the sort the Russians themselves used, especially since his own personal hygiene had presumably suffered from months of wilderness travel. The following excerpt is his account of the episode:

---

"While at Sitka I took a bath, which might be a very good thing to those that like it. On entering the building, I was much oppressed by the steam and heat, while an ill-looking, long-legged, stark-naked fellow was waiting to officiate as master of ceremonies. Having undressed in an ante-chamber, so far as decency would permit, I made my way into the bath-room, which was heated almost to suffocation. Having thus got me into his power, the gaunt attendant threw some water on the iron furnace, while, to avoid, as far as possible, the clouds of steam that were thus raised, I squatted myself down on the floor, perspiring profusely at every pore. I next seated myself on a bench, while bucket after bucket of hot water was thrown on my head; and then, making me stretch myself out, my tormentor soaped me all over from head to foot, rubbing and lathering me with a handful of pine tops. Once more taking his bucket, the horrid operator kept drenching me, the successive pailfuls descending gradually from nearly a boiling heat to the temperature of fifty degrees. The whole process occupied about an hour. I then returned to the ante-chamber, where, after being dried with hot towels, I was very glad to put on my clothes. It was impossible, however, to make my escape immediately, for I was so relaxed as to be obliged to recline on a sofa for a quarter of an hour; and then I withdrew, inwardly resolved never to undergo such another castigation."

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# Guest Editorial

## EPINEPHRINE FOR ANAPHYLAXIS

*On November 16, 1993, the American College of Allergy and Immunology recognized Claude A. Frazier, MD, with a special Award of Appreciation for his outstanding efforts in educating the public about the life-threatening dangers of insect allergy." Dr. Frazier is author of Insects and Allergy: and What to Do About Them (University of Oklahoma Press) and Insect Allergy: Allergic and Toxic Reactions to Insects and Other Arthropods (Warren H. Green Publishing). It is a pleasure to print the following editorial as he continues his crusade to save lives. Perhaps the Alaska State Medical Association should attempt to pass such a law in Alaska.*

The Editor

In my struggle as a lone crusader for a model bill to allow trained laymen to administer epinephrine to people having anaphylactic reactions I appeared before the FDA and the NIH Open Consensus Panel. I appeared before the AMA Board of Trustees at the request of my Senator from North Carolina. I began my talk, "I have some bad news for you and some good news. The bad news is that any one of you can walk out of this hotel and be stung and die in five minutes even if you have never had a previous reaction. The good news is that if a trained layman, such as a policeman, were allowed to give epinephrine, you would be saved."

After 10 years, many letters and frequent phone calls, The American Medical Association decided to prepare a model bill. This allows trained persons to administer epinephrine to a person suffering from a severe reaction to an insect sting. Death can occur in five to ten minutes and in most cases, this is insufficient time to get to a doctor or to a hospital and the person can die.

For 20 years, I have been a Lone Crusader in educating laymen, especially teachers, on the importance of diagnosis of an anaphylactic reaction due to insect stings and immediate treatment with an insect sting kit. Children playing at recess are very likely to get stung. If one has an anaphylactic reaction there usually is not time to get to a hospital or to a physician.

I feel so deeply about this lifesaving endeavor that I have done the work to date with no help financially. I have also been able to get the Army, Navy, Air Force, Marines, and Park Services to include insect sting kits containing epinephrine in their medical kits.

There are many needless deaths because of the ignorance of laymen. A nurse who was a patient of mine told me about her brother who was cutting a hedge around a doctor's home. He was stung and had an

anaphylactic reaction. He went inside and called for an ambulance. The doctor didn't know what to do, the EMT workers didn't know what to do, and the doctors at the emergency room didn't know what to do. He died. There are many needless deaths because of the ignorance in laymen.

Some high-school football players who die suddenly have their deaths in most cases attributed to heart attack. Investigations of some of these deaths have disclosed other players had heard the victim say that he had been stung. And the findings are consistent with anaphylaxis.

Sometimes a physician may be ignorant of proper management and administer antihistamines or steroids. For example, a young boy received multiple stings and was rushed to a nearby hospital. The physician wasn't sure of the proper treatment and sent him to another hospital. At neither hospital was the boy given epinephrine.

Early treatment with epinephrine after an anaphylactic reaction can be life saving. Even after this injection, the patient should always go to a hospital.

Persons in charge of others, especially outdoors, such as schoolteachers, coaches, school nurses, tennis or golf pros, and forest rangers should be legally allowed to administer lifesaving epinephrine.

The American Academy of Allergy and the American College of Allergy have passed special resolutions supporting this. Also, the past presidents of the American Academy of Family Practice and of the American Academy of Pediatrics have written letters of support.

Only 15 states have passed this law. To be legal in the administration of epinephrine, a layman must receive training by a physician. I have prepared a training program complete with slides that I can furnish at cost to physicians wishing to conduct training programs in states that have passed the law.

Recently, a lady from Florida called to tell me of her handicapped son who was severely allergic to bees. He had had a reaction and was given an insect sting kit. She explained this to the boy's bus driver. The driver told her that due to state law, he was unable to give any type injection or medication. The mother and I have been working to have Florida change this so even bus drivers can legally administer epinephrine. She asked me to appear before the Senate Subcommittee of Health Care and Health Rehabilitations Services.

In Asheville, a mother called me to report that her son, who also had had a previous reaction, was fishing one day when stung and died. She called because she knew of the work I was doing and told me the doctor had

given the boy antihistamines to take if stung again, and asked if this was right. I knew it was too late to save the boy's life, so I didn't say anything.

Dr. Donald Cook, of the American Academy of Pediatrics and School Health Committee wrote in a letter to the chairman of the Communications and Public Information Committee of the American Academy of Pediatrics, "the problem is this: (1) very few school people, parents or physicians are aware of the potential seriousness of the problem. (2) If they were aware, they wouldn't know what to do. (3) If they did know what to do they would be afraid to do it or (4) their school administrators would prevent them from doing it for fear of a law suit.

It is legal for a person having an insect sting reaction to obtain an insect sting kit but fatal reactions can and do occur in persons with no previous history of reaction. It still amazes me when I hear at lectures I give of a person who has had a severe reaction previously and was only given an antihistamine to carry. This will not save lives.

People do die from anaphylactic shock reactions to insect stings and I am convinced that there are many more not documented because of misdiagnosis.

Claude A. Frazier, MD  
Asheville, North Carolina.

(continued from page 182 — Interleukin-2)

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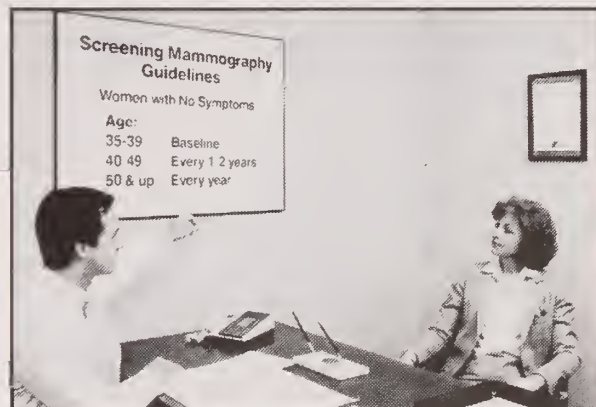
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